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SURGICAL MANAGEMENT OF COMPOUND DEPRESSED FRACTURE OF FRONTAL SINUS, CEREBROSPINAL RHINORRHEA AND PNEUMOCEPHALUS

E. S. GURDJIAN, M.D., AND J. E. WEBSTER, M.D.

DETROIT

The management of fracture of the frontal nus and its neighborhood, cerebrospinal rhinor- a and pneumocephalus have been discussed several authors. Among others, Teach- r,[1] Dandy,[2] Peet,[3] Rand,[4] Coleman,[5] Cairns [6] d Adson [7] have added much to our present day owledge of this subject. Certain phases of the atment are well worked out and generally ac- ted; other phases are in dispute. It has been r privilege to see many examples of fracture the frontal sinus at the Receiving and the ace Hospitals in Detroit. A portion of this perience was reported in 1932 (Gurdjian and awan [8]). During the past twelve years (March 30 to March 1942) we have treated 31 patients ith compound depressed fracture of the frontal nus and its neighborhood, 13 with cerebrospinal norrhea and 9 with pneumocephalus. In this per the lessons learned from the management these conditions will be discussed.

From the Department of Surgery, Wayne University llege of Medicine, and Grace Hospital.

Read at the meeting of the Middle Section of the erican Laryngological, Rhinological and Otological ciety, Detroit, Jan. 20, 1943.

1. Teachenor, F. R.: (*a*) Intracranial Complica- ns of Fracture of the Skull Involving Frontal Sinus, A. M. A. **88**:987 (March 26) 1927; (*b*) Pneumo- triele of the Cerebrum, Ann. Surg. **78**:561, 1923.

2. Dandy, W. E.: Pneumocephalus, Arch. Surg. :949 (May) 1926.

3. Peet, M. M.: Diagnosis of Acute Cranial and racranial Injuries, New York State J. Med. **28**:555, 28.

4. Rand, C. W.: Traumatic Pneumocephalus, Arch. rg. **20**:935 (June) 1930.

5. Coleman, C. C.: Fracture of the Skull Involving e Paranasal Sinuses and Mastoids, J. A. M. A. **109**: 13 (Nov. 13) 1937.

6. Cairns, H.: Injuries of the Frontal and Eth- idal Sinuses with Reference to Cerebrospinal Rhinor- a and Aerocele, J. Laryng. & Otol. **52**:589, 1937.

7. Adson, A. W.: (*a*) Cerebrospinal Rhinorrhea ssociated with Pneumocephalus, Pneumococcic Menin- is and Brain Abscess, Proc. Staff Meet., Mayo Clin. :746, 1941; (*b*) Cerebrospinal Rhinorrhea, Ann. Surg. 4:697, 1941.

8. Gurdjian, E. S., and Shawan, H. K.: Manage- nt of Skull Fracture Involving the Frontal Sinus, n. Surg. **95**:27, 1932.

It is generally accepted that simple fractures in the region of the frontal sinus should be treated conservatively except in the cases with cerebrospinal rhinorrhea, pneumocephalus or both, in which operative intervention may be preferable. Laceration over a nondepressed fracture in this neighborhood is also treated by the most conservative method, namely, cleansing and debriding of the wound and repair. Compound depressed fracture of the region of the frontal sinus is treated by operation after careful roentgen studies. Most authors extend the original laceration in order to get access to the area of damage (Teachenor,[1] Peet,[3] Rand [4]). Some have suggested a frontotemporal skin flap for exposure and repair of the depressed fracture (Cairns,[6] Rowbotham [9]). The management of pneumocephalus and cerebrospinal rhinorrhea may be surgical or conservative, depending on the acuteness of the condition and the progress of the patient. These various problems will be discussed more thoroughly under the proper headings.

COMPOUND DEPRESSED FRACTURE OF THE FRONTAL SINUS AND NEIGHBORHOOD

Compound depressed fracture of the frontal sinus and its neighborhood may establish a communication between the nose and the cranial cavity. Hence, it is a potential source of infection, and the rationale of operative intervention is to repair the communication between the intracranial cavity and the nasal spaces and thus to obviate the possible contamination of the former. Certain pertinent data on the 31 cases in which there was surgical treatment are grouped in table 1, and the cases will be discussed in greater detail in the next few paragraphs under the headings (1) causation, (2) clinical evidence of injury of the brain, (3) dural tears, (4) cerebrospinal fluid rhinorrhea, (5) pneumocephalus, (6) infection, (7) mortality and (8) management and surgical technic.

9. Rowbotham, G. F.: Acute Injuries of the Head, Baltimore, Williams & Wilkins Company, 1942, p. 176.

Causation.—The fracture in 2 cases was due to bullet wounds. In 3 cases a history of injury by a direct blow on the forehead was obtained. In 24 cases an automobile accident was the cause of the pathologic condition, and in 2 a fall was the etiologic factor.

Clinical Evidences of Injury of Brain.—In these patients, unlike those with fractures in posterior portions of the head, clinical evidences of injury of the brain, such as unconsciousness, convulsions, paralyses and other focal phenomena, were less common. Seven patients had no history of unconsciousness; 13 gave a history of unconsciousness of short duration, and 11 had severe clinical evidences of injury of the brain. Only a few among the latter group were moribund. The great majority were conscious soon after the injury and were sufficiently cooperative

TABLE 1.—*Data on Thirty-One Cases with Surgical Treatment of Compound Depressed Fractures Through Frontal Sinus, Orbit and Cribriform Plate*

	No. of Cases	
Patients recovered	28	
Patients died	3	
1. Clinical evidences of injury of the brain		
Slight	7	
Definite	13	
Severe	11	
2. Dural tears		
Present	21	
Absent	10	
3. Complications		
Cerebrospinal rhinorrhea	4	(no death)
Pneumocephalus	3	(no death)
Abscess of the brain	1	(no death)
Local infection	3	(no death)
Meningitis	3	(1 death)

to have the repair performed under local anesthesia. Although there may be little or no clinical evidence of injury of the brain, this does not preclude the possibility of dural tears, with destroyed and necrotic brain tissue. Many among the group who were conscious at the time of operation had extensive damage to the dural lining and the frontal pole of one hemisphere of the cerebrum or both.

Dural Tears.—Twenty-one persons in the group had dural tears, varying from small punctures to extensive lacerations. In fully half of these persons there was underlying lacerated and necrotic brain tissue. It was possible in almost all of the cases to dissect the dura from the base of the anterior fossa until the end of the dural laceration was reached. In many cases the laceration was sutured. In a few only a partial approximation could be obtained but the packing of the area with antiseptic iodoform gauze or gauze treated with metaphen in oil, according to the method of Peet,[3] was successful in obtaini complete repair.

Cerebrospinal Rhinorrhea.—This complicati occurred in 4 cases. In 2 the rhinorrhea dis peared after operation. In 1 there was esca not only of cerebrospinal fluid but of destroy and necrotic brain tissue from the left nostril. 1 case (case 7, cerebrospinal rhinorrhea grou the cerebrospinal rhinorrhea continued after t operative repair. In this case meningitis dev oped, which was successfully treated with sulfo amide drugs and which was followed by comple stoppage of the rhinorrhea. In 1 patient amo the 4 the rhinorrhea had developed seven and half weeks after repair of the compound d pressed fracture. In this case no tears of t dura were noted at the operation. This subje will be more thoroughly discussed under t heading of traumatic cerebrospinal rhinorrhea.

Pneumocephalus.—This complication was o served in 3 cases in this group. The presen of air in the cranial cavity with a compound simple fracture of the region of the frontal sin is an irrefutable argument in favor of the pre ence of a traumatic fistula between the outsi and the subarachnoid spaces of the cranial cavit

Infection.—Although the primary purpose the operative treatment in these cases is to o viate intracranial infection, infection may occ as a result of (1) a prolonged interval between t accident and the repair, (2) a high degree virulence of the offending organism or (3) lo ered resistance of the host. In this group the was 1 case of abscess of the brain, which w treated successfully by opening the wound an allowing the purulent matter to drain. The were 3 instances of local infection of the woun with no death. There were 3 cases of meningiti in 2 of which the patients recovered with chem therapy. The patient that died had been treate before 1935 and did not have the benefit of the apy with sulfonamide compounds.

At the present time we use prophylactic chem therapy with a sulfonamide compound before an after operative repair. The drug is used oral or subcutaneously, and in the last 4 cases sulfa pyridine was given for seven days, 90 to 12 grains (6 to 8 Gm) daily. Determinations of th levels of the drug in the blood and frequent bloo counts and urinalyses were made. In the last cases the wounds healed by primary intentio but these instances are too few to permit conclu sions concerning the value of the addition of the apy with sulfonamide drugs to the surgical man agement in such cases.

Mortality.—Three in this group of 31 patient died, 2 as a result of associated damage to th

in and 1 as a result of meningitis. Although emotherapeutic agents are available, careful erative management of compound depressed ctures of the region of the frontal sinus is ndatory. Surgical treatment can do much in viating intracranial infection and preventing current contamination. Patients with traumatic rebrospinal rhinorrhea still succumb to intra- anial infection in spite of competent use of emotherapy. Such therapy may control infec- n, but it does not help to heal cranionasal mmunications.

Surgical Technic.—Compound depressed frac- res in the region of the frontal sinuses should receive surgical treatment as soon as the condition of the patient permits. Careful roentgen examination should precede every operative repair, so that the extent of bony damage may be carefully evaluated before operation. We prefer exposing the lesion by extending the initial laceration in an appropriate manner. In some cases a frontotemporal skin flap is made. The comminuted pieces of bone are removed from the outer wall of the frontal sinus. The interior of this sinus is then carefully inspected. Lines of fracture and depression of the posterior wall are noted, and if comminuted and depressed pieces are present they are removed and the dural lining of the frontal lobe is exposed. Tears of the dura are repaired with silk (fig. 1). A tear extending posteriorly should be followed to its termination by careful dissection. At times the entire frontal sinus may be filled with pulped brain tissue. The necrotic tissue is sucked away. If the tear in the dura is beyond repair, the edges are approximated and held together with sutures and the area is packed with antiseptic gauze to hold the dura away from bony attachments. The pack may be removed slowly over a period of six to eight days, or the wound may be closed completely and six to eight days later reopened for removal of the gauze pack. Holding the dura away from bone is felt to be important in preventing a fistulous tract from developing between the cranial and the sinus cavity. A transplant of fascia lata may be used for extensive tears of the dura (Rand,[4] Cairns,[6] Campbell, Howard and Weary,[10] Gissane and Rank [11]).

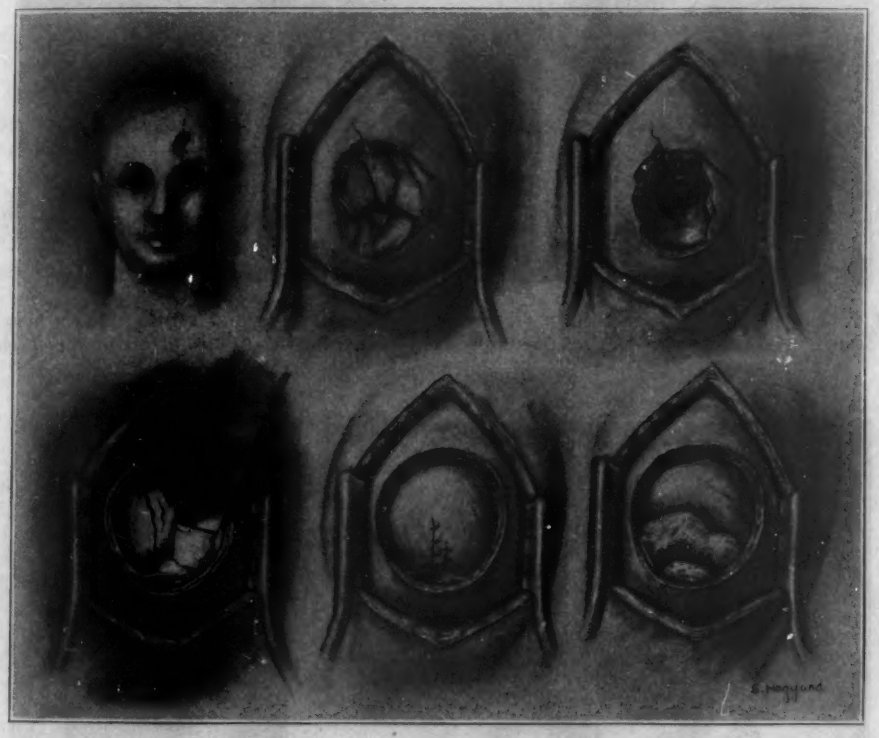

Fig. 1.—Operative sketch of a compound depressed fracture of the frontal sinus. The initial cut has been extended, and the area of depression exposed. The fragments of bone have been removed from the outer wall of the frontal sinus. After removal of the mucous membrane, the inner wall was inspected and found to be badly fragmented. The fragments of bone were removed, and two tears in the dura were exposed, which were repaired with interrupted silk sutures. The dura was then held away from bony attachments with an antiseptic gauze pack, which was removed on the fifth or sixth day.

10. Campbell, E.; Howard, W. P., and Weary, W. B.: Gunshot Wounds of the Brain, Arch. Surg. **44**:789 (May) 1942.

11. Gissane, W., and Rank, B. K.: Post-Traumatic Cerebrospinal Rhinorrhea with Case Report, Brit. J. Surg. **27**:717, 1940.

Careful work necessitates good exposure. Such exposure in this region may produce undesirable defects of the forehead, and these may need later operative correction. If the wound heals by primary intention and without local infection, the repair of the defect may be undertaken about four to six months after the accident. If postoperative local infection occurs, the defect may be repaired eight to ten months after the infection has completely cleared up. For small defects an autogenous osteoperiosteal transplant may be obtained with a trephine from the outer table of the skull (figs. 2 and 3). If the defect is large, an osteoperiosteal transplant of larger size may be obtained from the outer table of the skull, or a previously prepared metallic or plastic transplant may be used. Lately we have used a plastic material called "plexiglas," which seems promising for good and permanent repair. An excellent result can be achieved by the preoperative building of a mold of the defect, from which a plastic positive can be constructed to eliminate the defect of the forehead, orbital m gin and nasal region with accuracy.

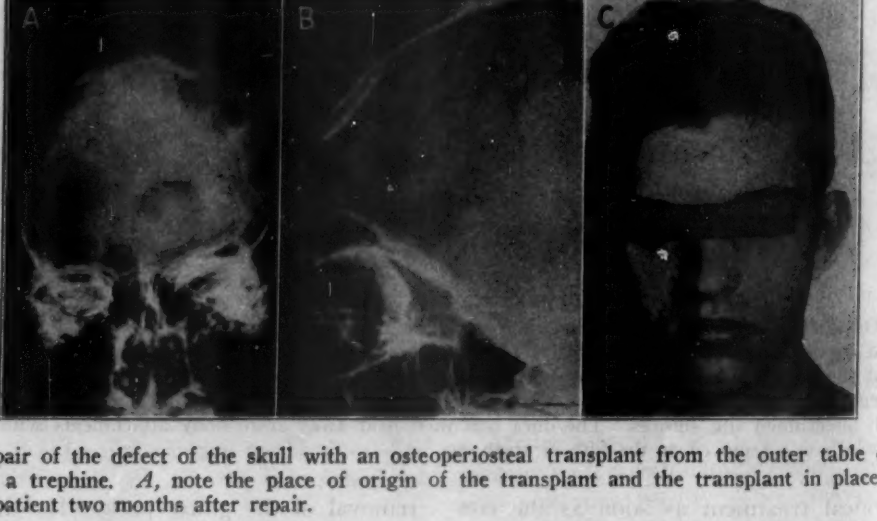

Fig. 2.—*A*, compound depressed fracture of the neighborhood of the frontal sinus, showing torn dura and lacerated left frontal lobe; *B*, the opening in the skull after the operation; *C*, the defect of the forehead.

Fig. 3.—Repair of the defect of the skull with an osteoperiosteal transplant from the outer table of the skul obtained with a trephine. *A*, note the place of origin of the transplant and the transplant in place; *B*, latera view; *C*, the patient two months after repair.

TRAUMATIC CEREBROSPINAL RHINORRHEA

Cerebrospinal rhinorrhea may be traumatic spontaneous. In this paper we are interested the traumatic type. The problem has been d cussed in many contributions, but most of th have been based on a report of a single case, w the exception of the papers by Dandy,[2] Ran Cairns[6] and Adson.[7] Adson reported 4 ca with surgical treatment, and Cairns discussed cases of cerebrospinal rhinorrhea and injury o paranasal sinuses. A review of the literature re veals that some authors favor conservative an others operative treatment. The present day management of such cases is somewhat simpler because of the success with the recent chemotherapeutic agents. However, traumatic cerebrospinal rhinorrhea is a serious complication of in jury of the head even with the availability o chemotherapeutic drugs. It is probably more

mmon than it is observed or described to be. the leakage may not be detected because of e patient's prone position in bed. In such es healing may occur during treatment with t in bed without detection of the complication. e condition is serious because infection may ter the cranial cavity from the nasal passages. eningitis and abscess of the brain are common quelae.

Healing of acute cerebrospinal rhinorrhea may ke place during absolute rest in bed, with no appearance of leakage. When cerebrospinal inorrhea recurs on one or more occasions, osure of the fistulous tract without operative tervention is rare and meningitis or abscess of e brain or both may develop several months or en years after the initial trauma. In such cases e disease is difficult to treat and frequently ends tally. The 13 cases of traumatic cerebrospinal inorrhea reported in this paper will be briefly scussed. They are divided into two groups, ose in which the condition was observed at the me of the acute injury of the head and those which recurrent cerebrospinal rhinorrhea was served several months or even years after the ute injury.

CASES OF CEREBROSPINAL RHINORRHEA OBSERVED AT THE TIME OF THE ACUTE INJURY OF THE HEAD

Case 1.—C. H., a man aged 32, entered the Receiving Hospital in March 1933. His head had been severely injured in an automobile accident, with fracture of the region of the frontal sinus and the upper jaw. He was unconscious on admission to the hospital and remained in a serious condition for six days. There was bleeding from the right side of the nose, and within twelve to eighteen hours the nasal discharge became clear. This lasted intermittently for two days and was never seen after that. The patient was too ill for surgical treatment. He slowly recovered and was discharged from the hospital in four weeks. He was observed for two years after the accident and had no complaints referable to nasal discharge.

Here is an example of cerebrospinal rhinorrhea which disappeared spontaneously. Roentgenograms revealed a fracture of the right frontal bone and of nasal and facial bones.

Case 2.—W. J. C., a man aged 41, was admitted to the Grace Hospital on June 30, 1935 and was discharged on July 21. The day before admission he was in an automobile accident. He was unconscious for a short time and was taken to the village jail and remained there overnight. During the night he noticed a discharge of clear fluid from the right nostril. The following day he was hospitalized at Grace Hospital, and roentgen examination showed a fracture of the right frontal bone involving the inner table and extending toward the cribriform plate. There were evidences of pneumocephalus with subdural collection of air (fig. 14 *C*). The patient was treated by strict rest in bed. Cerebrospinal rhinorrhea did not recur. He was observed for three years after the accident, and he had no recurrences.

This is another example of cerebrospinal rhinorrhea which disappeared spontaneously. The presence of air in the cranial cavity suggests a cranionasal fistula.

Case 3.—H. W., a man aged 37, entered the Delray General Hospital on July 29, 1939 and was discharged September 5. The patient had fallen from one deck to the next on a ship, a distance of 15 feet (4.5 meters). He was brought to the hospital in an unconscious state. There was copious discharge of clear fluid from the right nostril in the admitting room. He was treated by rest in bed for four weeks. Roentgen examination of the skull revealed a fracture of the right frontal bone involving the posterior wall of the frontal sinus and the right cribriform plate, with no depression. There have been no recurrences in two years.

This is another example of cerebrospinal rhinorrhea which was terminated by rest in bed.

Case 4.—J. B., a man aged 33, entered the Receiving Hospital in an unconscious state following an automobile accident on March 31, 1931. He was conscious and fairly normal the following day. While moving about in bed he noticed a discharge of clear fluid from the left nostril. This continued for several days, and frequently there was a gush of fluid. Roentgen examination of the skull showed no fracture. On the fifth day nuchal rigidity developed. There were evidences of pneumococcic meningitis on examination of the spinal fluid. He died two days later. At postmortem examination a half-inch (1.3 cm.) tear in the dura overlying the roof of the sphenoid sinus to the left of the midline and a hardly perceptible half-inch fracture involving the roof of the left sphenoid were observed.

This is an instance of cerebrospinal rhinorrhea with no roentgenologic evidence of fracture in which a fatal termination was caused by infection. It would have been most difficult to repair the tear in this region (over the sphenoid sinus), where dura and bone are separable only with the greatest difficulty.

Case 5.—E. K., a man aged 36, was admitted to Grace Hospital on Jan. 25, 1938 and was discharged on February 23. The patient entered the hospital in an unconscious state following an automobile accident. He remained disoriented for about a week. While roentgenograms were being taken with the patient's face down and his chin flexed, there was a sudden gush of clear fluid from the left nostril. This gush was repeated if one placed the patient's head down. Roentgen examination revealed a ragged line of fracture of the left frontal bone, involving the inner wall of the frontal sinus and extending toward the cribriform plate. A small amount of subdural air was noted in the frontal region (fig. 13*C*). It was thought that there was comminution of fragments at the base of the skull. The patient was operated on on January 27, and a frontal bone flap was turned (fig. 3′). The dura was gently lifted off the base of the anterior fossa. The line of the frontal fracture was followed along the posterior wall of the frontal sinus. Another line of fracture crossed the former obliquely at about the cribriform plate, where a spicule of bone was found embedded in the dura. This piece of bone, about 2 cm. long and 2

mm. wide, was removed, and thus a small tear in the dura was exposed. This defect was repaired with silk, and the dura was kept off the base of the skull with antiseptic gauze. The pack was removed six days later. The patient made an uneventful recovery. There has been no recurrence of cerebrospinal rhinorrhea up to the present time.

CASE 6.—W. S., a man aged 26, entered the Grace Hospital on April 21, 1940, after an automobile accident, and was discharged on May 9. There was a compound depressed fracture of the region of the frontal sinus, and clear fluid and brain tissue were discharged from the left nostril (fig. 4 *A* and *B*). This is one of the cases of compound depressed fracture of the frontal sinus reported in the previous portion of this paper. Roentgen examination showed depression and comminution of both tables of the frontal sinus, and at operation it was found to be filled with necrotic brain tissue. The inner wall of the frontal sinus was removed with care, and a jagged laceration of the dura with exuding brain tissue was exposed. The necrotic part of the brain was sucked away. The dura was sutured and was packed aw from the bone with antiseptic gauze. The pack w removed the sixth day, and the wound healed primary intention. On September 4 the patient ente the Grace Hospital for repair of the defect of the sk This was done by use of an osteoperiosteal transpl taken from the outer layer of the skull with a trephi Photographs (fig. 4 *C* and *D*) taken on Dec. 16, 19 show a satisfactory result.

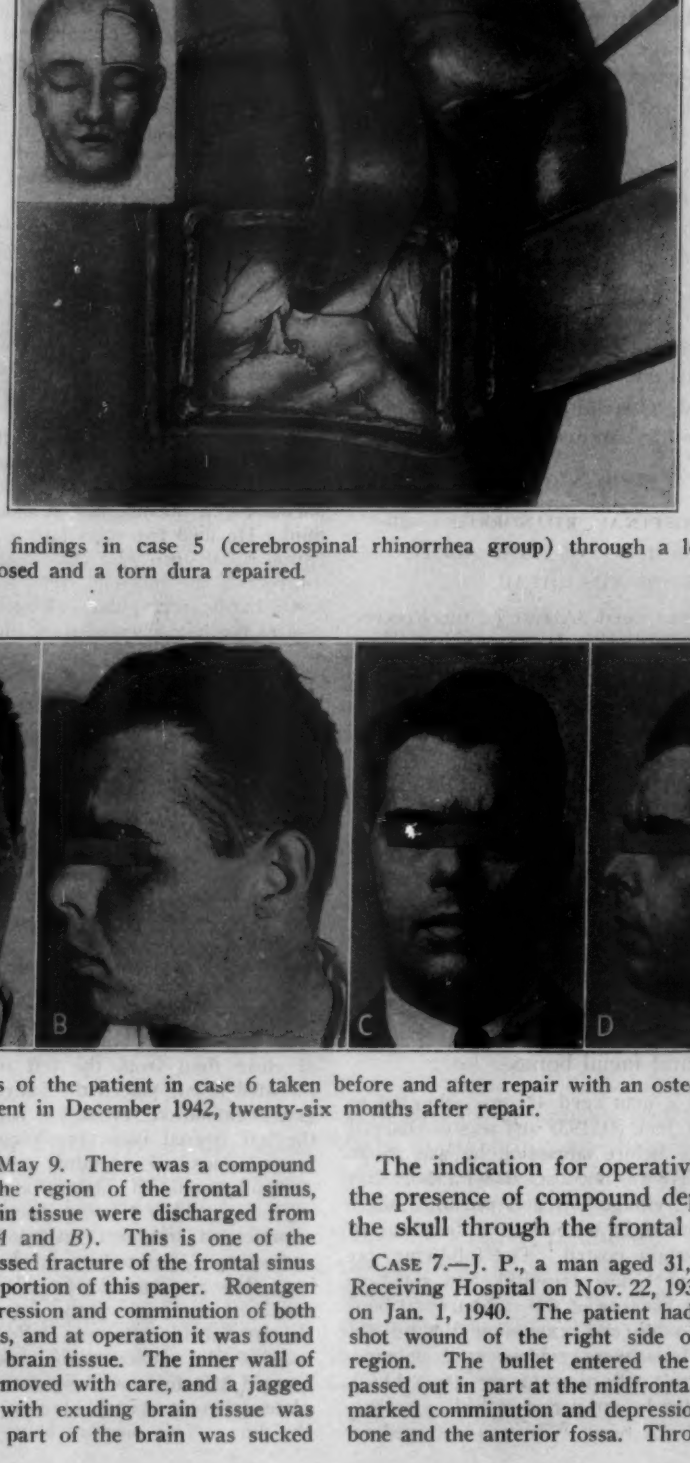

Fig. 3′.—Operative findings in case 5 (cerebrospinal rhinorrhea group) through a low frontal flap. Th anterior fossa was exposed and a torn dura repaired.

Fig. 4.—Photographs of the patient in case 6 taken before and after repair with an osteoperiosteal transplant *C* and *D* show the patient in December 1942, twenty-six months after repair.

The indication for operative intervention wa the presence of compound depressed fracture o the skull through the frontal sinus.

CASE 7.—J. P., a man aged 31, was admitted to th Receiving Hospital on Nov. 22, 1939 and was discharge on Jan. 1, 1940. The patient had a self-inflicted gun shot wound of the right side of the temporofronta region. The bullet entered the temporal bone an passed out in part at the midfrontal region. There wer marked comminution and depression of the right fronta bone and the anterior fossa. Through a frontotempora

in flap the frontal region was exposed. Many pieces bone had to be discarded. There was pulping of brain. About two thirds of the frontal lobe had be removed, and a badly comminuted anterior fossa as exposed. The lacerated dura was repaired with lk. The dura of the anterior fossa was dissected ay from the bone, and a small pack was used to hold away from the frontal sinuses. The wound was sed in layers. The pack was removed on the fifth d sixth days. There was rhinorrhea of cerebrospinal uid on the patient's entrance as well as for several ys after the accident. A roentgen examination five ys after repair revealed ventricular pneumocephalus g. 14*A*). A week after the injury rigidity of the neck veloped. Spinal puncture revealed turbid fluid with 800 pus cells. The meningitis was successfully treated ith sulfanilamide. The patient left the hospital on n. 1, 1940, with no nasal discharge, and was observed til June 1941, during this time having had no escape cerebrospinal fluid from the nose.

dicular incision was added in the midline. The sinuses were found to be badly shattered. After removing the fragments of bone, one could see both frontal poles with the covering dura. There were tears of the dura at the base of the skull with exuded brain tissue. The crista galli was badly shattered. The tears in the dura were sutured. The region was packed with antiseptic gauze, and the incision was closed in layers. The pack was removed on the fifth day. The patient made an uneventful recovery, and because of the rather narrow frontal sinuses no further repair work was necessary for cosmetic purposes (fig. 5). This patient was seen for about six months with no complaints of nasal discharge.

Here, again, the indication for operation was the presence of a compound depressed fracture of the skull through the frontal sinus. The complicating cerebrospinal fluid rhinorrhea was

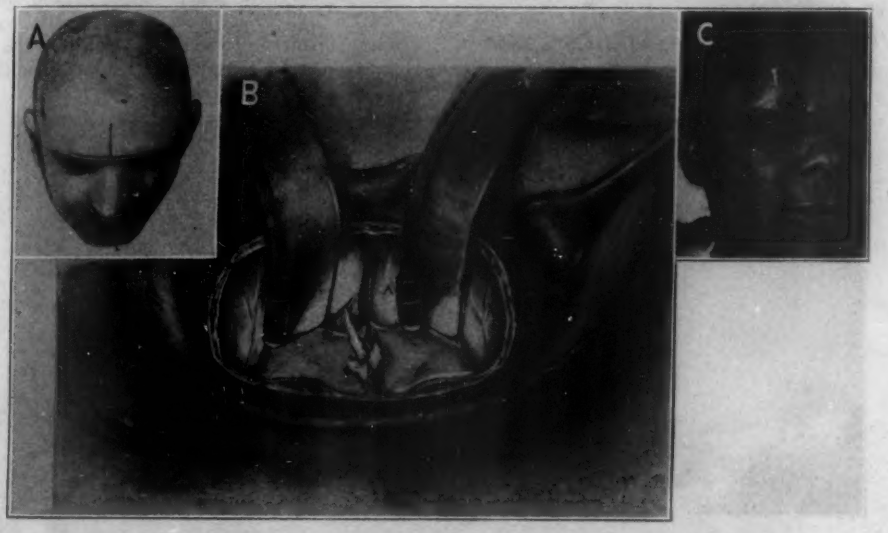

Fig. 5.—*A*, this patient (case 8, cerebrospinal rhinorrhea group) had a compound fracture of the frontal sinus and cribriform plate. *B*, the laceration from brow to brow accounts for the exposure as seen in the diagram. *C*, the patient before discharge from the hospital.

A complicating meningitis occurred in this case even though a repair had been effected. However, there was greater likelihood of good healing with operative repair than without. The continuation of the rhinorrhea may be explained on the basis either of incomplete healing or of breakdown of the repair. Both of these factors may have been present in this case. Repair of the defect of the skull was done at a later date. This case is also included in those of compound depressed fracture of the frontal sinus reported earlier in this paper.

Case 8.—E. B., a woman aged 36, entered the Receiving Hospital on Dec. 4, 1939 and was discharged on December 18. She was conscious on entrance. There was a laceration from brow to brow about 2 inches (5 cm.) long. The outer and inner walls of the frontal sinuses were depressed. Clear fluid escaped from the wound as well as from the right nostril. The initial laceration was extended over both brows. A perpen-

stopped by the operative repair. In cases 6, 7 and 8 operative treatment undoubtedly obviated possible later complications.

LATE AND RECURRENT CEREBROSPINAL RHINORRHEA

In the following 5 cases there was a history of recurrent cerebrospinal rhinorrhea due to trauma. One notes from reading the histories that results are tragic. Abscess of the brain and meningitis are common complications. In spite of use of chemotherapy and surgical treatment such conditions end fatally if allowed to go too far before radical surgical therapy is employed.

Case 9.—H. G., a boy aged 9 years, injured his head in November 1932 in a fall, in which a piece of wood was pushed up the left nostril with profuse bleeding. Three days later he had weakness of the right half of the body with aphasia. He was hospitalized, and meningoencephalitis developed, but he recovered. There

was intermittent cerebrospinal rhinorrhea from the left nostril from the time of the accident to his death. On one occasion the rhinorrhea was absent for five months. In May 1933 he was hospitalized at Grace Hospital for repair of the cranionasal fistula. Permission for operation was refused. He continued having attacks of cerebrospinal rhinorrhea with no other complaints. On July 9, 1935 he entered Harper Hospital in an unconscious state and died within forty-eight hours. At autopsy, there was observed a large, "goose egg-sized" abscess at the base of both frontal lobes. A patent communication existed between the cranial and the nasal cavities to the left of the midline and through the cribriform plate.

This case is an example of recurrent cerebrospinal rhinorrhea due to trauma with disastrous consequences. There was ample time for repair of the cranionasal fistula. It is interesting to note that there was complete cessation of rhinorrhea for five months.

Case 10.—D. J., a boy aged 14, fell from a railroad viaduct and injured his head in October 1935. The skull was fractured. He recovered except for the presence of cerebrospinal rhinorrhea from the right nostril. This continued with short remissions. In 1936 meningitis developed, which was treated successfully in Herman Keifer Hospital. The nasal drainage continued intermittently until March 31, 1938, when the boy was found unconscious on the floor in his room. The night before, he had appeared well. Examination showed that he had exophthalmos of the right eye, chemosis, stiffness of the neck and a positive Kernig sign. The optic disks were hyperemic. The spinal fluid was cloudy, having 20,000 polymorphonuclear cells per cubic millimeter. The infection was caused by type III pneumococci. Having been treated with antipneumococcus serum and sulfanilamide, the patient improved so remarkably that on April 21 the spinal fluid contained 24 cells with 8 polymorphonuclears. On April 20 he became mentally sluggish, complained constantly of headaches and vomited. Bilateral choked disks were noted. On April 29 a large abscess of the right frontal lobe was evacuated. The abscess contained live organisms. Injection of colloidal thorium dioxide revealed a patent opening into the nasal cavity and frontal sinus (fig. 6). The patient continued to improve for several days and then gradually failed. On May 25 he died. At autopsy it was revealed that the abscess of the fro lobe had been well drained, but a subdural abs 100 cc. in volume in the occipital and posterior pari region on the right side was seen. There were s necrosis and involvement of the underlying brain ti

This is another example of recurrent trauma cerebrospinal rhinorrhea with fatal ending. The were two attacks of meningitis. Although w appropriate chemotherapeutic agents a given fection may be treated with success, more rec rences are possible as long as the craniona fistula is patent. It is important to note t cerebrospinal rhinorrhea may be asymptomat its presence giving the patient a false securi The case just reported furnishes a good exam of this possibility.

Case 11.—B. G., a boy aged 13, was admitted to Grace Hospital on March 28, 1941 and was discharg on April 19. Ten weeks before, the patient fell that a long piece of steel was driven up the right nost He was hospitalized at St. Joseph's Hospital in Ponti Mich., for a few days. Intermittent discharge of cle fluid from the left nostril was noted, and this continue for six weeks. Concurrently with the stoppage of th cerebral rhinorrhea there developed increasing hea aches, nausea, vomiting and eventual stupor. The righ pupil was slightly larger than the left. There wer bilateral choked disks. Weakness of the right side o the lower part of the face, increased reflexes in th right half of the body and a Babinski sign on the righ side were noted. The neck was rigid. The spinal flui contained 440 white blood cells with 72 per cent poly morphonuclears. The pressure of the spinal fluid wa over 300 mm. of water. A diagnosis of abscess of th left frontal lobe was made. Through openings on either side of the midline the frontal lobes were explored, an purulent matter was obtained from the left side. Th abscess contained staphylococci. The ventricles an the subarachnoid spaces were washed with a 0.2 pe cent solution of sulfathiazole on four different occa sions. The fluid was injected into the ventricles an allowed to drain out from a lumbar puncture needle Between 400 and 600 cc. of fluid was used each time The patient was given sulfathiazole orally, and the level of the drug in the blood varied from 1.6 to 5.3 mg. per hundred cubic centimeters. Through a flap in the left

Fig. 6.—Injection of colloidal thorium dioxide into an abscess of the frontal lobe (case 10), showing the com munication between the abscess and the nasal cavity.

tal bone the base of the anterior fossa was explored durally and in the region of the cribriform plate. brain and the dura were found adherent, and the tionship was not upset, as it was thought that ing by adhesions had taken place. The patient's valescence was satisfactory. He was given several sfusions. On April 19 he was discharged in excel- condition.

He reentered the hospital June 4, 1941. Two weeks ore admission he began to feel nauseated. Four s prior to admission he had recurrence of the cere- spinal rhinorrhea. The spinal fluid had 1,300 white od cells with 85 per cent polymorphonuclears. Cul- e showed Staphylococcus albus. Another operation performed on June 9, but the patient died two days r operation.

management may be. It also demonstrates the roles of both chemotherapy and surgical treatment. Reinfection through a fistulous tract was fatal in spite of chemotherapy.

CASE 12.—J. W., a man aged 35, a physician, entered the Grace Hospital on Dec. 25, 1934, having sustained a compound depressed fracture of the skull through the right frontal sinus. The initial laceration was extended. Both tables of the frontal sinus were found depressed and comminuted. These fragments were removed. The dura was carefully inspected for tears, and none were discovered. The wound was packed with antiseptic gauze and closed in layers. Seven days later the pack was removed. The wound healed satisfactorily, and the patient was discharged twelve days after the injury.

Fig. 7 (case 13, cerebrospinal rhinorrhea group).—*A*, anteroposterior view of a fracture of the left frontal d nasal bones; *B*, lateral view; *C* and *D*, encephalograms taken before the patient's discharge from the hospital.

It would have been much simpler to operate this patient before the formation of the ab- ess of the frontal lobe. At that time the anionasal fistula could have been repaired. The fort made to expose the fistula after the drain- e of the abscess was commendable, but the inion that healing by adhesion had occurred ould be criticized. Certainly much could have en gained by proper exposure and repair of e opening, particularly since therapy with sulf- namide compounds could have been used to ht a potential reinfection. Experience in this se demonstrates how trying the problem of

Seven and a half weeks after the accident there was serous discharge from the right nostril without other complaints. After many consultations the patient decided on conservative therapy. He was then placed in bed with no privileges for six weeks. There has been no reappearance of the discharge of cerebrospinal fluid in eight years.

Here is another example of delayed cerebrospinal rhinorrhea with recovery by means of rest in bed. If the rhinorrhea had recurred, the patient would have been strongly advised to undergo operative repair of the cranionasal fistula.

CASE 13.—J. G., a man aged 52, was in an automobile accident in September 1941. He had an extensive simple

linear fracture of the left side of the frontoparietal region and the left side of the upper jaw. He had bloody spinal fluid and remained semiconscious and disoriented for two weeks. Because of his deranged mental state it was thought advisable to obtain encephalograms (fig. 7). These were made on September 23 and were essentially normal. He then went home and carried on satisfactorily until January 1942, when a high fever and rigidity of the neck developed. The examination of the spinal fluid revealed pneumococcic meningitis. Under therapy with a sulfonamide compound he improved. Although the spinal fluid cleared up completely, the patient continued in a confused state. Roentgen examination showed pneumocephalus (fig. 8). Air injected from below revealed the cerebral aerocele to be communicating with the ventricle. It had partially destroyed the left frontal lobe. When the patient was placed with his head down there was discharge of

apparently carried on satisfactorily until men gitis developed seven weeks after he left hospital. With chemotherapy he recovered fr the meningitis, but because of mental stupor a bizarre neurologic findings a roentgenogram w obtained, which showed pneumocephalus. T left frontal lobe was now cystic, whereas ni weeks before encephalograms had showed no s lesion. At the time of the second cerebral pne mographic study, cerebrospinal rhinorrhea w first noted. The aerocele of the left frontal l was found to communicate with the lateral ve tricle. The cystic degeneration of the left fron lobe was probably brought about as follows (W

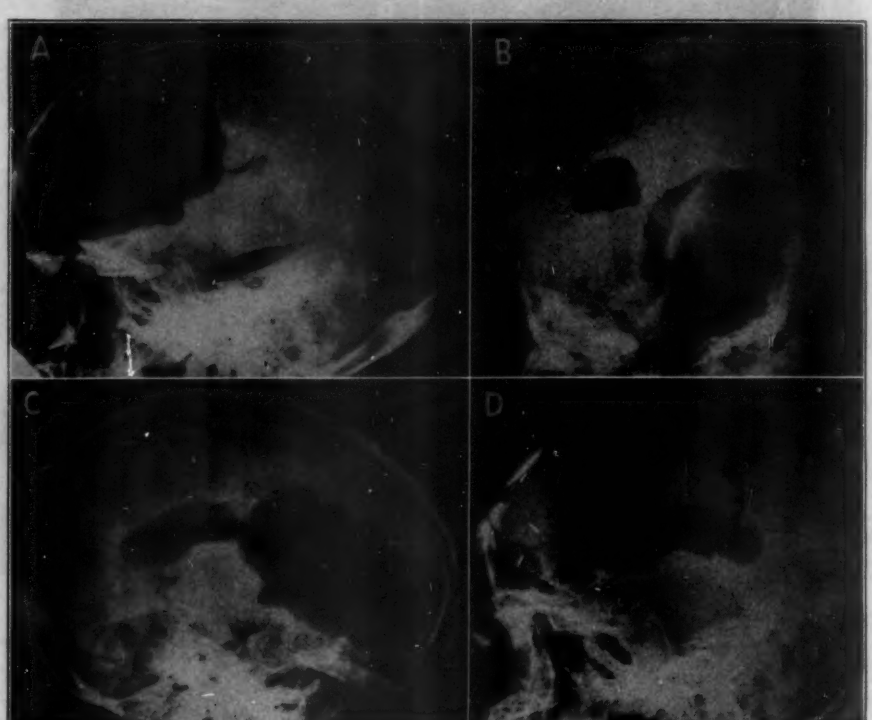

Fig. 8 (case 13, cerebrospinal rhinorrhea group).—*A*, pneumocephalus of the left frontal lobe; *B* and *C*, th position of the brain cyst in relation to the ventricles; *D*, view after repair of the cranionasal fistula, showin absence of air in the cranial cavity.

cerebrospinal fluid from the left nostril. The fistula causing the cerebrospinal rhinorrhea was then attacked through a bone flap in the left frontoparietal region, both extradurally and intradurally. There was a neat hole about 5 mm. in diameter just to the left of the midline and over the midportion of the ethmoid labyrinth. An oval flap of falx cerebri was used to repair the hole in the dura (fig. 9). The attachment of the flap of the falx to the base of the skull was left intact. The patient made conspicuous improvement from that time on. There was eventual improvement of mental capacity, and he now appears to be normal (fig. 10). There has been no reappearance of cerebrospinal rhinorrhea.

Because of his abnormal mental status, encephalograms were obtained after the acute injury and just before the patient was discharged. He

darcz,[12] Duken [13]). Initially, the orbital surfac of the left frontal lobe was probably bruised, wit adhesions between the pia-arachnoid and the dur in the vicinity of the cranionasal fistula. Wit sudden increase in the intracranial pressure fro such acts as coughing and sneezing, air may hav entered the cranial cavity and dissected a progres sively enlarging space in the frontal lobe becaus of "ball valve" action of the fistula. It is likel

12. Wodarcz, A.: Zur Kasuistik der intracraniell Pneumatozele, München. med. Wchnschr. **62**:868, 191

13. Duken, J.: Ueber zwei Falle von intrakraniell Pneumatocele nach Schussverletzung, München. me Wchnschr. **62**:598, 1915.

t this patient would not have survived without
e aid of chemotherapy. But it is also hoped
t use of surgical treatment has obviated the
currence of the infection.

due to tumor of the brain, with thinning and eventual rupture of the neighborhood of the cribriform plate or erosion of the anterior fossa by tumor. Internal hydrocephalus of childhood may

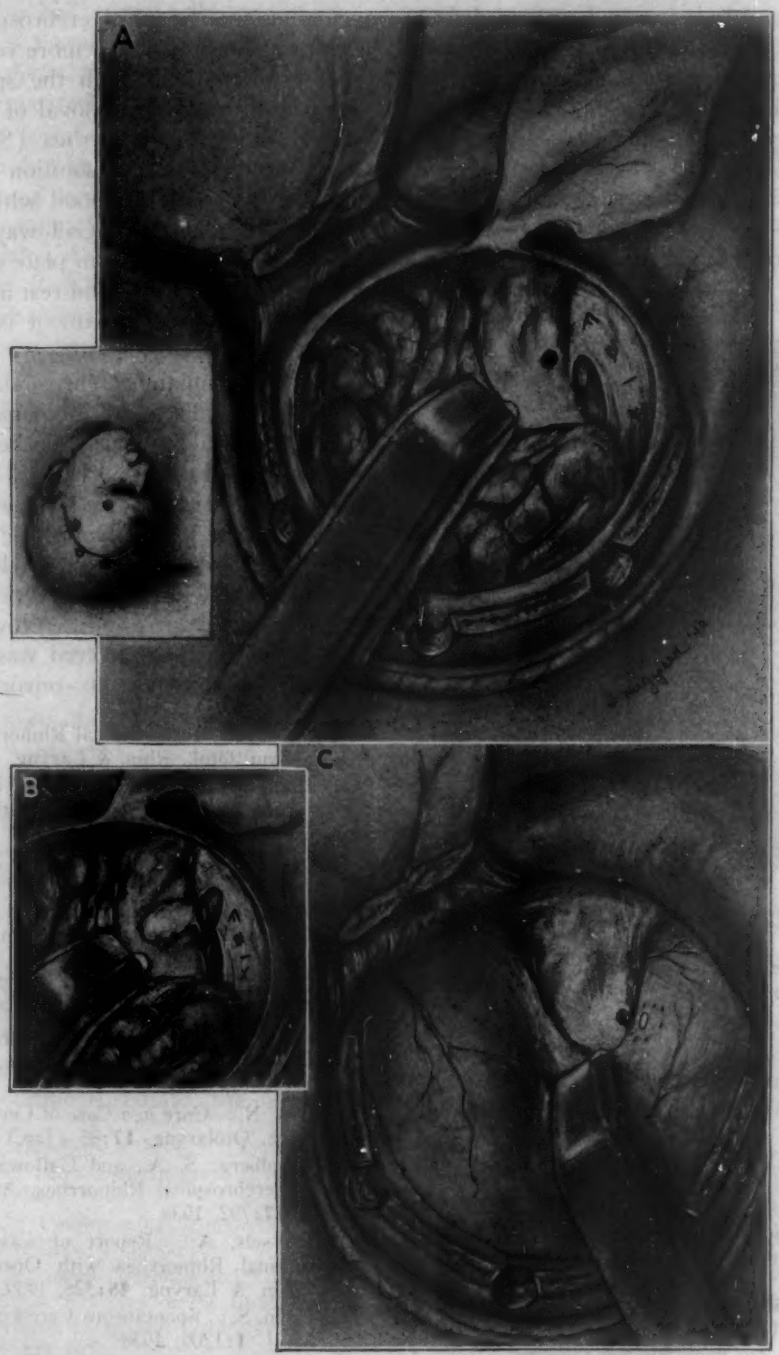

Fig. 9.—Repair of the cranionasal fistula of the patient in case 13 with the use of a piece of falx. The position of the hole can be seen in *A*. *B* shows the sutured reflexion of the falx, and *C* presents the extradural appearance of the repair.

COMMENT

Cerebrospinal rhinorrhea may be spontaneous or traumatic. Instances of the spontaneous type include cases with increased intracranial pressure be eventually associated with cerebrospinal rhinorrhea by thinning and rupture of the neighborhood of the cribriform plate. Meningoceles of varying size (usually minute) in the neighbor-

hood of the exit of olfactory nerves may extend into the nasal cavity with the external appearance of polypi. Erosion of the roof of the nasal cavity by tumor (Friedman,[14] Hill,[15] Graham [16]) or infection (Chiari [17]) may eventuate in cerebrospinal rhinorrhea. In a few instances of definite clinically proved cases of spontaneous cerebrospinal rhinorrhea no cranionasal communications have been found. In these cases there may have been minute meningoceles about the exit of the olfactory nerves. The so-called spontaneous variety is only slightly less serious than the traumatic type. Locke,[18] who reviewed 14 cases of spontaneous cerebrospinal rhinorrhea from the literature with 12 satisfactory autopsies, found

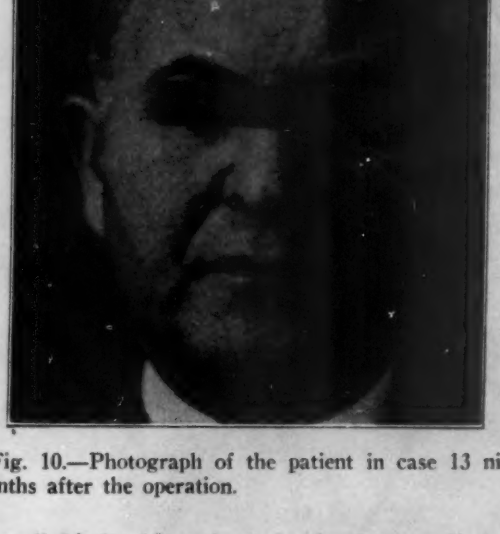

Fig. 10.—Photograph of the patient in case 13 nine months after the operation.

that all of the 12 patients in these cases died of meningitis. More recently, the patients of Som and Kramer,[19] Weil and Womack,[20] Shea [21] and Plunkett and Lendrum [22] died of intracranial infection. On the other hand, Plum [23] described a case of eighteen years' duration. In Collet's case the disease had a course of three years. Feinblatt and Damrau [25] reported an instance of recurrent spontaneous cerebrospinal rhinorrhea with remissions of two or more years. The treatment given patients with the spontaneous type of infection included removal of a pituitary cyst with cessation of rhinorrhea (Smith and Walter [26]), application of a solution of silver nitrate to the fistular neighborhood with apparent cure (Fox,[27] Friedberg and Galloway [28]), placing a muscle over the cribriform plate with cessation of discharge (Wessels [29]) and rest in bed (Livin [30]). As a rule, the best treatment is to find and to remove the underlying cause.

Cases of traumatic cerebrospinal rhinorrhea include those of discharge following a nasal operation or external injury to the head. Postoperative cerebrospinal rhinorrhea is serious. Several cases have been reported since the review by Campbell [31] in 1928. Donnelly,[32] Higbee,[33] Jervey [34] and Cairns [6] also reported such cases. In all we have collected 15 cases, in which 5 patients died from meningitis and 9 recovered. Included among those who recovered was 1 patient with meningitis. Spontaneous repair of the cranio-

14. Friedman, A. P.: Tumor hypophysis und Liquorrhea nasalis, Arch. f. Psychiat. **88**:359, 1929.

15. Hill, F. T.: Nasopharyngeal Fibroma with Report of a Case with Secondary Pneumocephalus, Ann. Otol., Rhin. & Laryng. **51**:105, 1942.

16. Graham, T. O.: Cerebrospinal Rhinorrhea, J. Laryng. & Otol. **52**:344, 1937.

17. Chiari, H.: Ueber einen Fall von Luftansammlung in den Ventrikeln des menschlichen Gehirns, Ztschr. f. Heilk. **5**:383, 1884.

18. Locke, C. E., Jr.: The Spontaneous Escape of Cerebrospinal Fluid Through the Nose: Its Occurrence with Brain Tumor, Arch. Neurol. & Psychiat. **15**:309 (March) 1926.

19. Som, M. L., and Kramer, R.: Cerebrospinal Rhinorrhea Pathological Findings, Laryngoscope **50**: 1167, 1940.

20. Weil, A. I., and Womack, D. R.: Cerebrospinal Rhinorrhea, Laryngoscope **43**:767, 1933.

21. Shea, J.: Cerebrospinal Rhinorrhea with Autopsy Report, Ann. Otol., Rhin. & Laryng. **47**:253, 1938.

22. Plunkett, J. E., and Lendrum, F. C.: A Case of Spontaneous Pneumocephalus and Cerebrospinal Rhinorrhea, Canad. M. A. J. **33**:512, 1935.

23. Plum, F. A.: Cerebrospinal Rhinorrhea, Arch. Otolaryng. **12**:84 (Jan.) 1931.

24. Collet, F. J.: Ecoulement nasal de liquide cephalorachidien prolonge pendant des annees, Rev. d'otoneuro-opht. **10**:660, 1932.

25. Feinblatt, H., and Damrau, F.: Cerebrospinal Rhinorrhea, M. Rec. **139**:324, 1934.

26. Smith, C., and Walter, L.: Cerebrospinal Rhinorrhea with Cyst of the Pituitary Body, Arch. Otolaryng. **14**:610 (Nov.) 1931.

27. Fox, N.: Cure in a Case of Cerebrospinal Rhinorrhea, Arch. Otolaryng. **17**:85 (Jan.) 1933.

28. Friedberg, S. A., and Galloway, T. C.: Spontaneous Cerebrospinal Rhinorrhea, Ann. Otol., Rhin. & Laryng. **47**:792, 1938.

29. Wessels, A.: Report of Case of Spontaneous Cerebrospinal Rhinorrhea with Operative Cure, Ann. Otol., Rhin. & Laryng. **48**:528, 1939.

30. Livin, S.: Spontaneous Cerebrospinal Rhinorrhea, Brit. M. J. **1**:1209, 1938.

31. Campbell, E. H.: Cerebrospinal Rhinorrhea Following Intra-Nasal Surgery, Ann. Otol., Rhin. & Laryng. **37**:865, 1928.

32. Donnelly, J. C.: Cerebrospinal Rhinorrhea, Arch. Otolaryng. **16**:350 (Sept.) 1932.

33. Higbee, D. R.: Notes on the Treatment of Cerebrospinal Rhinorrhea, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:521, 1933.

34. Jervey, J. W.: Cerebrospinal Rhinorrhea Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:431, 1932.

sal fistula took place in all but 2 cases. In the se reported by Bulson and Bulson [35] the cerebospinal rhinorrhea continued for six years with untoward effects. The patient in Wurster's [36] se had discharge of cerebrospinal fluid from the se for six and a half years, followed by complete stoppage of the discharge. At the time the author's report, the cerebrospinal rhinorhea had not reappeared for three years.

The traumatic form of cerebrospinal rhinorhea may be seen during the acute state of injury the head or as a late complication. The reurrent form of traumatic cerebrospinal rhinorhea is usually fatal if untreated. Teachenor,[1] and,[4] Lawson,[37] Taft,[38] Smith and Malcolmon [39] and Flower [40] have reported instances of erebrospinal rhinorrhea with fatal termination. t is possible that many more fatal cases are not eported in the literature.

The diagnosis of cerebrospinal rhinorrhea is ased on the history of trauma, intermittent and sually unilateral discharge of clear fluid with o associated infection of the respiratory tract nd chemical analysis of the fluid. The chemical nalysis reveals sugar, which is not present in asal secretions. A simple method of recognition the "handkerchief test." When a handkerchief used, the fabric does not stiffen after drying, hereas watery nasal discharge causes a stiffeng. Constantin [41] injected a solution of fluoescein into the spine and recovered it in the asal discharge in one hour and thirty minutes in case of cerebrospinal rhinorrhea. Fox [27] reorted a case in which a neutral dye was injected ntraspinally and recovered from the nose, causng a bluish tinge of the fluid discharge. A careful urvey of the nose by a rhinologist may reveal he point or region of leakage. If the fluid comes rom the roof of the nose, the cranionasal fistula probably through the cribriform plate; if it is rom below the middle turbinate, the fistula is probably through the frontal sinus. In 1 of the cases reported by Cairns,[6] the leakage of cerebrospinal fluid took place on the side opposite that of the fractured anterior fossa, because of the presence of a clot in the homolateral frontal sinus.

Of the 13 patients reported on here, 8 were seen in the acute stage of trauma to the head; 5 had recurrent cerebrospinal rhinorrhea. Of those seen during the acute phase, 4 patients had surgical treatment and the remaining 4 were treated conservatively. The indication for operation on 3 patients was the presence of a compound depressed fracture of the frontal sinus and neighborhood. The other patient receiving surgical treatment had a comminuted fracture of the inner wall of the frontal sinus and of the cribriform plate with no lacerations of the forehead. The indication for such treatment in this patient's case was evident, and the observations at operation included a tear of the dura and a spicule of bone driven into it (case 5).

TABLE 2.—*Analysis of Thirteen Cases of Traumatic Cerebrospinal Rhinorrhea*

	No. of Cases
Acute:	
Patients recovered	8
Patients died	1
Associated pneumocephalus	3
No pneumocephalus	6
Operative treatment	4
Patients recovered	4
Conservative treatment	5
Patients recovered	4
Recurrent:	
Patients recovered	2
Patients died	3

Among the patients with acute cerebrospinal rhinorrhea not having surgical treatment, 1 was too ill for such treatment to be considered. However, over a period of several days he recovered with no reappearance of the nasal discharge. The other 3 were carefully studied. In 1 roentgen examination showed no fracture. The remaining 2 had linear fractures at the base of the anterior fossa. The rhinorrhea occurred only once and could not be induced by varying maneuvers in the 2 patients with fracture, but the patient with no roentgenologic evidence of fracture (case 4) had repeated attacks of cerebrospinal rhinorrhea. Meningitis developed on the fifth day, and he died two days later. The other 2 recovered with no recurrence for two or more years.

Certain pertinent conclusions may be drawn from the foregoing analysis (table 2). Cerebrospinal rhinorrhea may be treated conservatively in the absence of compound depressed fracture of the frontal sinus and neighborhood, in the absence

35. Bulson, A. E., and Bulson, E. L.: Cerebrospinal Rhinorrhea Following Intranasal Surgery, J. A. M. A. **0**:1969 (Dec. 21) 1929.

36. Wurster, H. C.: Cerebrospinal Rhinorrhea, J. Indiana M. A. **30**:199, 1937.

37. Lawson, A.: A Case of Cerebrospinal Rhinorrhea Following on a Multiple Fracture of the Skull Which Involved the Left Frontal Sinus and Orbit, Tr. Ophth. Soc. U. Kingdom **54**:307, 1934.

38. Taft, R. B.: An Unusual Type of Traumatic Pneumocephalus, Am. J. Roentgenol. **25**:800, 1931.

39. Smith, J. S., and Malcolmson, P. H.: Traumatic Pneumocephalus, Canad. M. A. J. **30**:650, 1937.

40. Flower, C. F.: Pneumocephalus, U. S. Nav. M. Bull. **39**:553, 1941.

41. Constantin, P. M.: Ecoulement spontane de liquide cephalo-rachidien par les fosses nasales, Bull. Soc. oto-rhino-laryng. **18**:205, 1919.

of extensive and progressive pneumocephalus and in the absence of comminuted fragments at the base of the anterior fossa. If the rhinorrhea recurs after a few days of strict rest in bed, arrangements must be considered for operative intervention. If the rhinorrhea recurs after several weeks of rest in bed, then there is a patent cranionasal fistula which probably would never heal completely without operative repair. Such a patient should receive surgical treatment before complicating meningitis, abscess of the brain or both occur.

It is interesting that in a number of the cases of recurrent cerebrospinal rhinorrhea the discharge stopped for several weeks. In case 9 it stopped for five months on one occasion. In the reviewed literature there are many examples of spontaneous cerebrospinal rhinorrhea of long duration. In Plum's case [23] the duration of the condition was eighteen years. Collet [24] described a case in which the disease had a course of three years. Traumatic intermittent cerebrospinal rhinorrhea is usually of short duration, two or three years at the most. However, in a case described by Fribourg-Blanc, Lasalle and Germain [42] the patient had intermittent recurrent cerebrospinal rhinorrhea for seventeen years as a result of a war wound of the frontal region. In this series, in case 9 the patient lived for two years and eight months and in case 10 for two years and seven months before he died from complications. Frequently the rhinorrhea is not associated with such symptoms as headache and stupor, which may obtain if there is associated pneumocephalus. Many patients and their relatives consider the cerebrospinal rhinorrhea a mere nuisance so long as there is no pain. For this reason permission for operative intervention may be refused.

In these 5 cases of recurrent rhinorrhea, 3 patients died of abscess of the brain or meningitis or of both. In 2 cases there developed more than one attack of meningitis. This is important in that, although with chemotherapy, it may be possible to cure a given attack of meningitis, the patient may succumb to the second or third attack of infection so long as the cranionasal fistula is patent.

All 3 patients with recurrent cerebrospinal rhinorrhea who died had an adequate interval before the onset of the fatal complication, and the cranionasal communication could have been repaired with comparative safety.

There is 1 instance of spontaneous repair in the group with recurrent rhinorrhea. The patient has been followed for eight years without recurrences. In spite of this case, it is felt that recurrent cerebrospinal rhinorrhea means a nonhealing fistula, and the best method of treatment is operative, particularly since, with the aid of chemotherapy, the danger of infection from the operation is minimal.

MANAGEMENT AND TECHNIC

The treatment of cerebrospinal rhinorrhea may be conservative or operative or both. After careful consideration of the clinical findings, if conservative treatment is decided on, the patient is strictly confined to bed in a slight semi-Fowler position. He is asked to remain quiet and move about as little as possible. He is advised against blowing the nose. Prophylactic doses of a sulfonamide compound are justifiable.

If operative management is desirable the problem varies according to the type of condition. The three types encountered are: first, acute cerebrospinal rhinorrhea; second, recurrent cerebrospinal rhinorrhea, and, third, recurrent cerebrospinal rhinorrhea with complications (meningitis, abscess of the brain or pneumocephalus).

In a case of acute cerebrospinal rhinorrhea the indication for operation may be the presence of a compound depressed fracture in the neighborhood of the frontal sinus or comminution and fragmentation of the inner wall of the frontal sinus with no laceration of the forehead. In a case of compound depressed fracture the initial wound may be enlarged by appropriate extension, the area of damage exposed and the torn dura repaired. If there are no lacerations or depressions and if the case is one of recurrent rhinorrhea with no complications, one may expose the anterior fossa by turning an adequate and low frontal flap. The torn dura may then be exposed externally and repaired. Both an extradural and an intradural approach may be desirable in those cases in which the cranionasal fistula is in the posterior portion of the cribriform plate and over the sphenoid sinus. If the dura about the hole can be mobilized, it may be possible to suture it. More frequently, a fascial transplant may be necessary to repair the fistula. Fascia lata (Dandy,[3] Randall,[5] Cairns [6]), temporal fascia, pericranial fascia (Trotter [43]), osteoperiosteal transplant from the tibia (Gissane and Rank [11]), cautery of the fistula and plugging it with transplant of muscle (Echols and Holcombe [44]) have been used. Stuck and

42. Fribourg-Blanc, O.; Lasalle, and Germain: Deux observations de pneumatocèle intracranienne, Rev. neurol. **2**:51, 1934.

43. Trotter, cited by Lawson.[37]

44. Echols, D. H., and Holcombe, R. G.: Traumatic Intracerebral Pneumatocele, South. Surgeon **10**:58 1941.

atherly [45] placed a piece of fascia lata over defect intradurally without suture. Their ient recovered with no nasal discharge. In of the reported cases we were able to use oval flap of falx cerebri to repair the hole in dura. The base of the flap was left attached the skull. Adson [7] suggested a bilateral frontal in order to afford adequate exposure. Prolactic use of sulfonamide drugs before and er operation is of great help.

The problem is difficult in the cases in which complicating condition is already present. If patient entered the hospital because of mengitis, abscess of the brain or both, he should treated for these conditions before repair of cranionasal fistula is considered. It is to be ped that such patients may be seen before inction has entered the cranial cavity, when reir may be undertaken with relative safety.

PNEUMOCEPHALUS

The subject of pneumocephalus should be considered in connection with that of cerebrospinal inorrhea and fracture about the paranasal uses because of intimate relationship of cause nd effect between the two. However, pneumophalus may be present without cerebrospinal inorrhea, and many patients with cerebrospinal inorrhea have no associated pneumocephalus. hiari [17] noted the presence of air in the cranial vity at the autopsy table in a case of purulent hmoiditis with erosion of bone in 1884. Luckett [46] reported on a case of traumatic pneumophalus proved by roentgen examination in 1913. ince then, many reports have appeared in the terature on the subject of traumatic pneumophalus. In a great many of the reported cases erebrospinal rhinorrhea and pneumocephalus or loody or cerebrospinal otorrhea and pneumophalus coexist. The contributions of Dandy [2] nd Rand [4] have laid the foundation for the raonale of operative treatment. Dandy's [2] excellent paper includes a comprehensive review of he literature. The review of Worms, Didiée nd Grumbach [47] is also excellent. Recently, Killian [48] collected 110 cases of pneumocephalus, including 1 of his own. In all but 18 the origin was traumatic. In 17 the pneumocephalus was caused by gunshot wounds; in 59 it was associated with fracture of the frontal bone, and in 16, with fracture of the petrous bone. His analysis suggested that conservative management is the most successful.

The presence of air in the cranial cavity of itself is no basis for concern except that, following trauma, it is due to the existence of a communication between the intracranial spaces and the outside air. Considered in this light, it is a dangerous sign. It may cause serious sequelae by, first, introduction of organisms and, second, continuous entrance of air into the brain, as this may result in signs and symptoms of compression, including stupor, convulsions, paralyses and aphasia.

REPORT OF CASES

Nine examples of pneumocephalus are reported in table 3. Air in the cranial cavity was associated with cerebrospinal rhinorrhea in 4 cases. These have already been described and are cases 2, 5, 7 and 13. The pneumocephalus in these cases was another evidence of patent cranionasal fistula. The remaining 5 cases include 4 of pneumocephalus associated with cerebrospinal or bloody otorrhea and 1 of compound depressed fracture of the midoccipital region. The pertinent points concerning the signs and symptoms in these cases will be presented in the next few paragraphs.

TABLE 3.—*Analysis of Nine Cases of Traumatic Pneumocephalus*

	No. of Cases
Patients recovered	7
Patients died	2
Portal of entry:	
Cerebrospinal rhinorrhea	4
Cerebrospinal otorrhea	4
Compound fracture of occipital bone	1
Operative treatment:	
Patients recovered	3
Patients died	1
Conservative treatment:	
Patients recovered	4
Patients died	1

CASE 1.—H. G., a man aged 55, entered the Grace Hospital on March 8, 1935 and was discharged on April 7. He was unconscious from an automobile accident and remained unconscious and disoriented for about ten days. At first there was bleeding from the right ear. The discharge became serosanguineous and then clear. There was paralysis of the right peripheral facial nerve. Roentgen examination revealed an extensive fracture of the temporal and mastoid bones with subdural pneumocephalus (fig. 11 *B*). Because of lowering

45. Stuck, R. M., and Weatherly, F. E.: Pneumocephalus, Arch. Neurol. & Psychiat. **44**:1093 (Nov.) 1940.

46. Luckett, W. H.: Air in the Ventricles of the Brain Following Fracture of the Skull, Surg., Gynec. & Obst. **17**:237, 1913.

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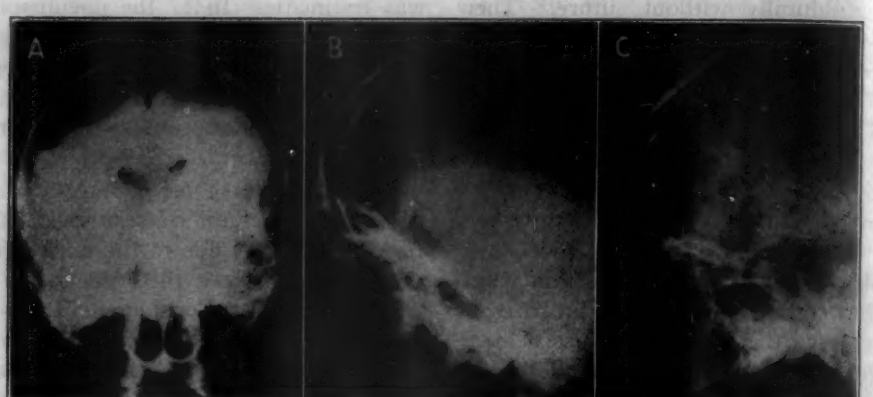

Fig. 11.—*A*, pneumocephalus associated with a bleeding right ear. Subdural accumulation with some vent ular collection. *B*, subdural pneumocephalus associated with discharge of cerebrospinal fluid from the right *C*, subdural accumulation of air with cerebrospinal otorrhea from the left ear.

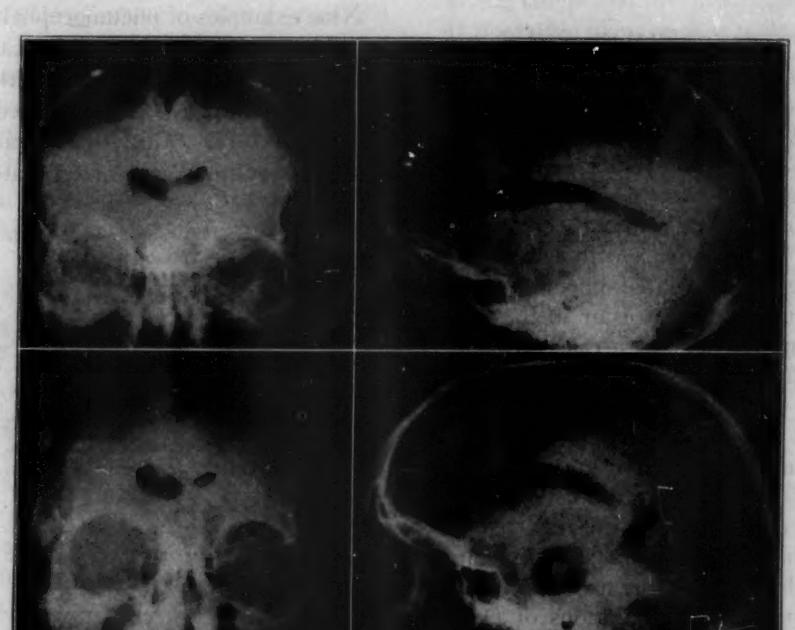

Fig. 12.—The same patient shown in figure 11 *A*. Roentgenograms show the progressive disappearance of a Each group of anteroposterior and lateral views were obtained three days apart.

rate and jacksonian convulsions of the right half body, an operation was performed on March 15. iddle meningeal hemorrhage on the right side and ubdural collection of spinal fluid on the left side e evacuated. Roentgen examination on March 10 shown marked reduction in the size of the pneumo-alus.

he patient's convalescence was stormy, but he left hospital in satisfactory condition. The paralysis of facial nerve showed some improvement in the fol-ing twelve months, and although voluntary control returned, there are spasms of the right side of the .

The pneumocephalus was treated conserva-ely with complete disappearance of the symp-ns.

neck, a positive Kernig sign, and a cloudy spinal fluid. Roentgen examination showed no air in the cranial cavity.

The patient's bodily activity may have contributed to the development of meningitis in this case. The presence of cerebrospinal rhinorrhea is interesting. The autopsy showed no fracture in the anterior fossa but a short longitudinal fracture of the right petrous bone; hence, it was reasonable for one to assume that the spinal fluid entered the nose from the right middle ear.

Case 3.—C. C., a man aged 52, entered the Receiving Hospital on Dec. 30, 1936 and was discharged on Jan. 22, 1937. He became unconscious after a fall down

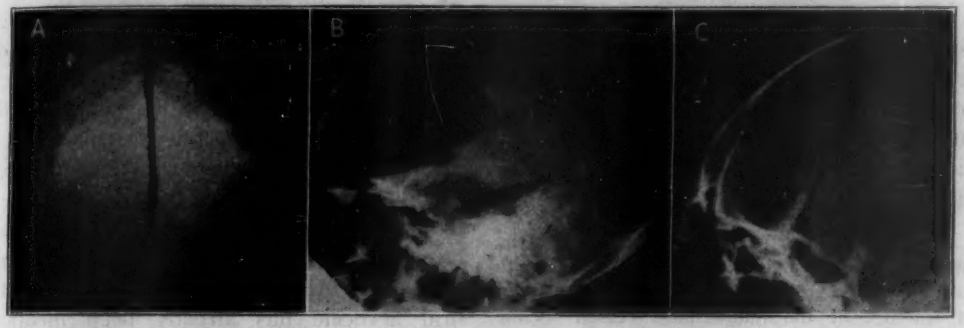

Fig. 13.—*A*, interhemispheric accumulation of air in a case of cerebrospinal otorrhea; *B*, pneumocephalus ociated with cerebrospinal rhinorrhea in case 13; *C*, pneumocephalus associated with cerebrospinal rhinorrhea case 5.

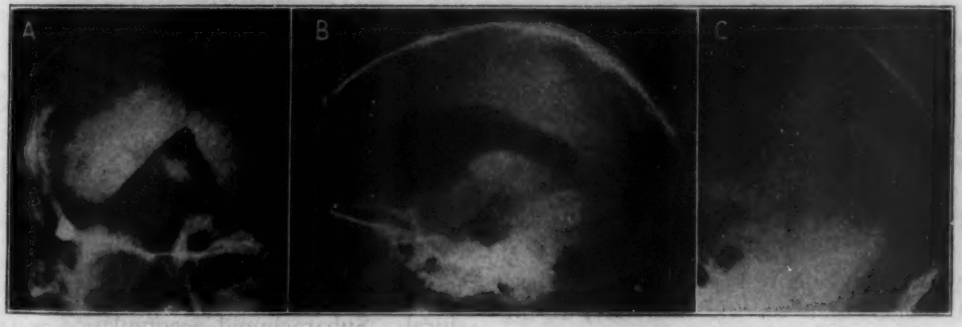

Fig. 14.—*A*, pneumocephalus associated with cerebrospinal rhinorrhea in case 7; *B*, pneumocephalus associated ith a compound depressed fracture of the midoccipital region; *C*, subdural pneumocephalus associated with cere-ospinal rhinorrhea in case 2.

Case 2.—J. S., a man aged 45, entered the Grace ospital on March 30, 1935 and was discharged against vice on April 6. He had fallen downstairs and had come unconscious for about a half-hour. There was eeding from the right ear followed by otorrhea of inal fluid during the stay in the hospital. On one casion there was cerebrospinal rhinorrhea on the ght side. It was thought that the spinal fluid entered e nose through the eustachian tube and its neighbor-od. Roentgen examination revealed subdural air in e frontal region (fig. 11 *C*), but no fractures could demonstrated even with a Stenvers projection. The atient left the hospital against advice on the seventh y. Two days after discharge he showed evidences meningitis. He was rehospitalized on April 9 but ed soon after entrance. There were rigidity of the cellar steps. There was bleeding from the left ear. He was conscious and cooperative twenty-four hours later. Roentgen examination revealed fracture of the region of the left temporal and mastoid bones with marked subdural and ventricular pneumocephalus (fig. 11 *A*). Roentgenograms were obtained every two days and showed progressive diminution of air, with complete disappearance on Jan. 13, 1937 (fig. 12). This patient was seen in the outpatient department for six months with no complaints suggesting recurrence of pneumocephalus.

This case is a good example of pneumocephalus successfully treated by conservative means.

Case 4.—J. C., a man aged 40, entered the Grace Hospital on Oct. 4, 1942 and was discharged on Octo-

ber 19 with orders to remain in bed at home for fifteen days more. After an automobile accident he was unconscious and semiconscious for three days. There was bleeding from the right ear, and he showed peripheral paralysis of the right facial nerve. Roentgen examination revealed linear fracture of the right temporal bone with pneumocephalus between the two hemispheres (fig. 13 *A*). Two days later there was less air in the cranial cavity. He was given 90 grains (6 Gm.) of sulfanilamide a day for eight days. The level of the drug in the blood rose to 6 mg. per hundred cubic centimeters. The patient returned to work on Dec. 15, but he still has paralysis of the right facial nerve.

The reports of these 4 cases demonstrate the propriety of conservative treatment in cases of cerebrospinal otorrhea. In case 2, in which the patient died, there was disappearance of air, and the infection cannot be attributed to the pneumocephalus any more than to the presence of cerebrospinal otorrhea. If a check roentgen examination shows increase in the air content, operative intervention may be advisable. This has not been true in our cases of bloody or cerebrospinal otorrhea.

CASE 5.—G. O., a girl aged 8 years, entered the Receiving Hospital in an unconscious state following an automobile accident on June 19, 1931. There were a depressed fracture of the midoccipital region to the right of the midline and a fracture of the right humerus. Spinal fluid discharged from the wound in the scalp. Roentgen examination on June 21 revealed a ventricular pneumocephalus (fig. 14 *B*). Because of the seriousness of her condition, she was not referred to the neurosurgical division until June 25. An operation was performed, the compound depressed fracture being repaired and the wound closed in layers. Unfortunately, the repair broke down in the ensuing two days, and concurrently there were evidences of encephalitis. There were jacksonian spells on the right side and then on the left side. The patient remained stuporous and had a temperature of 103 to 104 F. for several days. The spinal fluid remained remarkably clear. The child died on June 30.

If the compound depressed fracture had been repaired sooner than on the fifth day, results might have been different.

COMMENT

The air may enter the cranial cavity at the time of the acute trauma, but more frequently it enters a few days after the injury (Killian [48]). Pneumocephalus may appear many weeks or months after a severe injury of the head (Campbell, Howard and Weary,[10] Stuck and Weatherly,[45] Echols and Holcombe [44] and others). In such cases a complicating cerebrospinal rhinorrhea may be present. In a few the presence of air in the cranial cavity is the only sign of a communication between the intracranial space and the outside air. On the other hand, cerebrospinal rhinorrhea may be present without a complicating pneumocephalus. In the patient of Fribourg-Blanc, Lasalle and Germain,[42] pneumocephalus did not appear for seventeen years, though the patient's history suggested occasional cerebrospinal rhinorrhea (two or three times yearly).

The usual avenue of entrance of air is a cribronasal fistula. This is true in most of the cases of latent pneumocephalus. Fracture through the petrous bone, with bloody or cerebrospinal otorrhea, is the second important avenue of entrance. In 4 cases in our series pneumocephalus was associated with cerebrospinal rhinorrhea and in 2 cases with cerebrospinal or bloody otorrhea. In 1 case a compound depressed fracture of the occipital region which had been left untreated for five days was the avenue of entrance of air.

The entrance of air into the cranial cavity may be caused either by a sudden increase in the intranasal pressure, which forces in the air through an open or practically open fistula, or by an intracranial negative pressure caused by excessive leakage of cerebrospinal fluid through the patent fistula. However, cerebrospinal rhinorrhea may be present for many years without pneumocephalus. The mechanism of production of intracerebral aerocele has already been mentioned in connection with case 13 in the cerebrospinal rhinorrhea group. In the vicinity of the cribronasal fistula, adhesions may occur between the pia-arachnoid and the dura. When air is forced in by sneezing, coughing or blowing the nose, it dissects a progressively enlarging space in the brain, particularly if its escape is prevented by a "ball valve" action of the fistula neighborhood. With repeated entrance of air, the cerebral tissue is destroyed and replaced by a cyst cavity (Wodarcz,[13] Duken)[13] which may eventually invade the ventricular space.

The air may be subdural, ventricular, intracerebral or subarachnoid. Most commonly it is subdural. Subarachnoid accumulation is rare. In our group of 9 cases subdural collection of air was observed in 6, ventricular in 4 and intracerebral in 1.

The diagnosis of pneumocephalus is simple after roentgen examination. If conscious, the patient usually has severe headaches. If the onset of the pneumocephalus was sudden, the symptoms are those of severe headaches and collapse of bodily functions suggestive of shock. Accumulation of air in the subdural and subarachnoid spaces may cause localized pressure with focal phenomena. Hemiparesis, hemiplegia, aphasia and convulsions may be seen. The mental state of the patient is characteristically deranged. He is somnolent and stuporous. His reactions are poor, and his mental plane is markedly lowered

the patient with recurrent pneumocephalus, r a day or two of rest in bed there may be inite improvement in the mental processes, as t of the air is absorbed.

MANAGEMENT

In the management of pneumocephalus, the lowing questions are pertinent: 1. Where is fistula? 2. Is the fistulous communication l patent? 3. Is the pneumocephalus progres-e? The position of the fistula is important in t in certain regions there is greater likelihood contamination than in others. This is true th cranionasal fistulas. Communication be-een the intracranial cavity and the outside air rough the ear is less serious, and patients with rebrospinal or bloody otorrhea with pneumo-phalus can be treated conservatively with con-fence. Prophylactic therapy with sulfonamide mpound during the period of discharge seems rth while.

Conservative treatment, namely, strict rest in d in a semi-Fowler position, has a definite ace in the management of pneumocephalus as-ciated with acute injury of the head. Particu-rly with entrance of air through the temporal ne conservative treatment may be used with ccess. Frequent roentgen examinations may veal the status of the accumulation of air. An crease in this accumulation is always significant d may spell a need for operative intervention.

For the acute form of pneumocephalus opera-ve intervention may be indicated because of the esence of compound depressed fracture in the eighborhood of the frontal sinus or elsewhere. he presence of comminution of fragments in acture of the posterior wall of the frontal sinus ith no overlying laceration of the scalp may be dication for operation if complicated with cere-rospinal rhinorrhea, with or without pneumo-phalus. Operative intervention in such cases ay be preferable not merely because of the pres-nce of air in the cranial cavity but because of the mpound nature of the wound, the patent cra-ionasal fistula and the clinical progress of the atient.

The treatment of pneumocephalus which ap-ears several months or years after the acute in-ury is, in general, operative. Cranionasal fis-ula is the cause of latent pneumocephalus in ractically all of the cases. Its presence is not nly a constant source of contamination but an venue for repeated attacks of aerocele. An in-racerebral pneumocephalus is particularly dan-erous and should be treated by operative repair f the fistulous tract. In the cases of Campbell, Howard and Weary,[10] Stuck and Weatherly,[48] Cairns,[6] Gissane and Rank,[11] McKinney,[49] Miller, Klemmer and Snoke [50] patients were successfully treated by operation. On the other hand, Barden,[51] Bromberg,[52] Killian,[48] Boninger,[53] Lippens,[54] Peter,[55] Fribourg-Blanc, Lasalle and Germain,[42] and Campbell [56] stated that their patients recovered by conservative treatment. There seems little doubt that with present day availability of chemotherapeutic agents, operative repair is the best method of treatment.

In operative management the position of the fistula and the presence of a complicating cerebrospinal rhinorrhea are important. If the fistula extends through the anterior fossa, an adequate frontal bone flap may be used to explore this region both extradurally and intradurally. The tear in the dura may be repaired by suture and fascial or osteoperiosteal transplant. The problem is similar to that of operative management of recurrent cerebrospinal rhinorrhea.

CONCLUSIONS

1. A simple fracture in the frontal sinus and its neighborhood should be treated conservatively in the absence of complicating cerebrospinal rhinorrhea or pneumocephalus. A laceration over a nondepressed or comminuted fracture in this neighborhood is also treated by the most conservative method, namely, débridement and repair of the laceration.

2. A compound depressed fracture in the frontal sinus and its neighborhood is an operative problem. The operative rationale is to repair dural tears and thus obviate intracranial infection. Good exposure may be obtained by extension of the original laceration.

3. Cerebrospinal rhinorrhea associated with acute injury of the head may be treated con-

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53. Boninger, G.: Ventrikulographie durch Schadelbruch, Zentralbl. f. Chir. **67**:630, 1940.

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56. Campbell, D.: Pneumocephalus internus nach Schadelbruch, Ztschr. f. d. ges. Neurol. u. Psychiat. **119**:481, 1929.

servatively in some cases with success. If a compound depressed fracture of the neighborhood of the frontal sinus is present, operative repair is imperative. If roentgen examinations reveal comminution of fragments in the anterior fossa, operative repair of the cranionasal fistula is preferable if the patient's condition permits.

4. In a case of recurrent cerebrospinal rhinorrhea of traumatic origin there should be operative repair of the cranionasal fistula. Spontaneous repair in this group is infrequent. In the presence of complicating meningitis or abscess of the brain the latter should be treated first.

5. Pneumocephalus may be treated conservatively with success in some cases. Pneumocephalus due to cerebrospinal or bloody otorrhea be treated by rest in bed with little fear of in tion. Frequent roentgen investigations sh reveal any increase in the intracranial air. I increase is present, operative treatment ma advisable.

6. Pneumocephalus associated with cere spinal rhinorrhea is dangerous, and the treatm of choice is operative repair of the cranion fistula.

7. In pneumocephalus, irrespective of po of entrance, operative repair is indicated w subsequent roentgen examinations show that amount of intracranial air has increased.

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ANATOMIC VARIATIONS OF THE LATERAL AND SIGMOID SINUSES

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Anatomic variations of the sinuses of the dura ater, however infrequent, may present puzzling agnostic and operative problems in the presence thrombophlebitis. Some of the variations are xtremely rare. In this paper a hitherto unown anomaly of the sigmoid sinus will be ported and a short description of most of the blished observations which are important for e otologist will be given. In view of the isting differences in the nomenclature, in this per the horizontal portion of the transverse nus will be called the lateral sinus and the rtical portion will be called the sigmoid sinus.

REPORT OF A CASE

A 65 year old patient was operated on for septic rombophlebitis of the right transverse sinus, but the gmoid sinus could not be located; the patient died of rulent meningitis due to pneumococci of type III hich originated from a deep epidural abscess of the ght posterior fossa. Autopsy showed the following natomic features:

The left lateral and sigmoid sinuses were large, ended a large jugular bulb and produced a deep groove in e left occipital and temporal bones. The left superior nd inferior petrosal sinuses showed normal develop- ent. The right lateral sinus was narrow and barely dmitted a probe. The right sigmoid sinus could not e found and there was no evidence of a groove in e occipital and temporal bones. The superior petro- al sinus was normal and connected the narrow lateral nus with the cavernous sinus. The inferior petrosal nus was wider than usual, communicated regularly ith the cavernous sinus and laterally ended in a pouch hich occupied the place of the jugular bulb and opened nferiorly into a narrow internal jugular vein (fig. 1 *A*).

EMRYOLOGY

A short outline of the development of the inuses will help to explain the genesis of the ariations in question. The sinuses develop from hree venous plexuses, which in the primary type f circulation are drained by the "head vein" (Streeter [1]). The "head vein" runs alongside the brain tube. In early embryonic life the primitive anterior and middle venous plexuses of the brain drain mainly into the vena capitis lateralis, which leaves the endocranium through the spurious jugular foramen and drains into the system of the external jugular vein. The spurious jugular foramen is located above the jaw joint and is usually obliterated in fully developed skulls. The posterior venous plexuses drain into the internal jugular vein, which gives origin to the sigmoid sinus (fig. 1 *B*). Later the sigmoid sinus becomes connected with the anterior and middle plexuses by means of a horizontally running channel which represents the lateral sinus. The vena capitis lateralis is obliterated (Labbé [2]) (fig. 1 *C*).

In most mammals, both before and after birth, the venous blood of the brain is drained almost entirely through the spurious jugular foramen into the external jugular vein; this venous structure seems to be phylogenetically the older one. The internal jugular vein plays only a secondary role in the drainage. In man and in some anthropoid monkeys the main venous channel of the endocranium is the internal jugular vein.

An incomplete or missing connection between these two types of venous plexuses or persistence of the primitive venous channels explains most of the anomalies. A large number of persistent, patent spurious jugular foramens were reported by different authors: first by Krause,[3] in 1842, and then by Hyrtl,[4] Knott [5] (in 26 cases), Luschka,[6] Cheatle [7] (in 23 of 2,585 skulls examined) and others. This fact confirms the embryologic data and suggests a more frequent occurrence of the primitive, persistent petrosquamous sinus which passes through that foramen than is commonly assumed. Cheatle found 3 spurious jugular foramens located in the glenoid fossa of the temporomandibular joint, 3 in the zygomatic process, 6 at the base of the

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From the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital.

1. Streeter, G. L.: Contrib. Embryol. **8**:5, 1918.

2. Labbé, C.: Arch. de physiol. norm. et path. **1**:1, 1883.

3. Krause, C. F. T.: Handbuch der menschlichen Anatomie, ed. 2, Hannover, Hahn, 1842, p. 914.

4. Hyrtl: Wien. med. Wchnschr. **12**:289, 1862.

5. Knott, J. F.: J. Anat. & Physiol. **16**:27, 1881.

6. Luschka: Ztschr. f. rat. Med. **7**:72, 1859

7. Cheatle, A.: Tr. Internat. Cong. Otol., 1899, sect. 6, p. 160.

malar bone and 11 close to the glaserian fissure. It may be mentioned here that studies of the skulls alone may lead to faulty conclusions about the venous sinuses, because frequently a small sinus is located in a large groove of the skull or a large sinus in a small groove.

I found few or no data about anatomic variations of the venous sinuses in textbooks of anatomy. In the following paragraph many of these variations important to the otologist will be discussed:

1. A contracted jugular foramen with a small internal jugular vein was found by Linser [8] in 30 of 1,022 skulls examined. The contralateral jugular vein sometimes was four times as large in such cases. A contracted jugular foramen was five times more frequent on the left side.

Swift [9] reported a case in which both jug foramens of a newborn child were rudiment the infant died three days after birth.

2. Absence of the lateral sinus is a anomaly and usually occurs on the left side. few cases were reported by Lieutaud,[10] Halle and Furstenberg.[12] The superior petrosal si was present in these cases, and continued dire into the sigmoid sinus. This anomaly oc

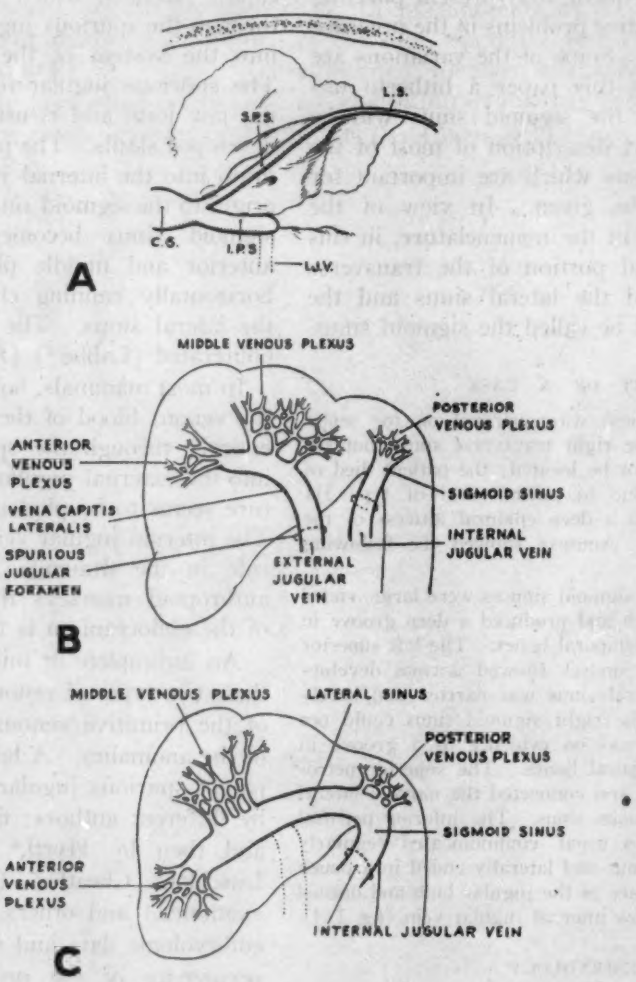

Fig. 1.—*A*, diagrammatic representation of the region of the sinuses of the patient in the case reported (intra cranial view). *C.S.*, cavernous sinus; *S.P.S.*, superior petrosal sinus; *I.P.S.*, inferior pertosal sinus; *L.S.* lateral sinus; *I.J.V.*, internal jugular vein. The right sigmoid sinus is absent. *B* and *C*, diagrammatic repre sentations of the region of the sinuses in early and in later embryonic life, respectively.

if the internal jugular vein is not connected or i only insufficiently united with the anterior an middle venous plexuses of the brain. The sig

8. Linser: Beitr. z. klin. Chir. **28**:642, 1900.

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sinus, which develops from the internal lar vein, is present, while the lateral sinus her absent or is filiform (fig. 2 *B*).

A small transverse sinus leaving the endoium through the mastoid foramen was obed by Knott [5] in 2 cases; the sinus was 1.5 in diameter (fig. 2 *C*[1]). In Hoople's [13] the lateral sinus measured 3 mm. and continued with the mastoid emissary vein just below the superior knee of the sigmoid sinus. A persistent petrosquamous sinus was present (fig. 2*C*[2]).

4. A normal lateral sinus leaving the skull through an enlarged mastoid foramen was reported by Malacarne [14]; it was a bilateral anomaly and the emissary foramen measured

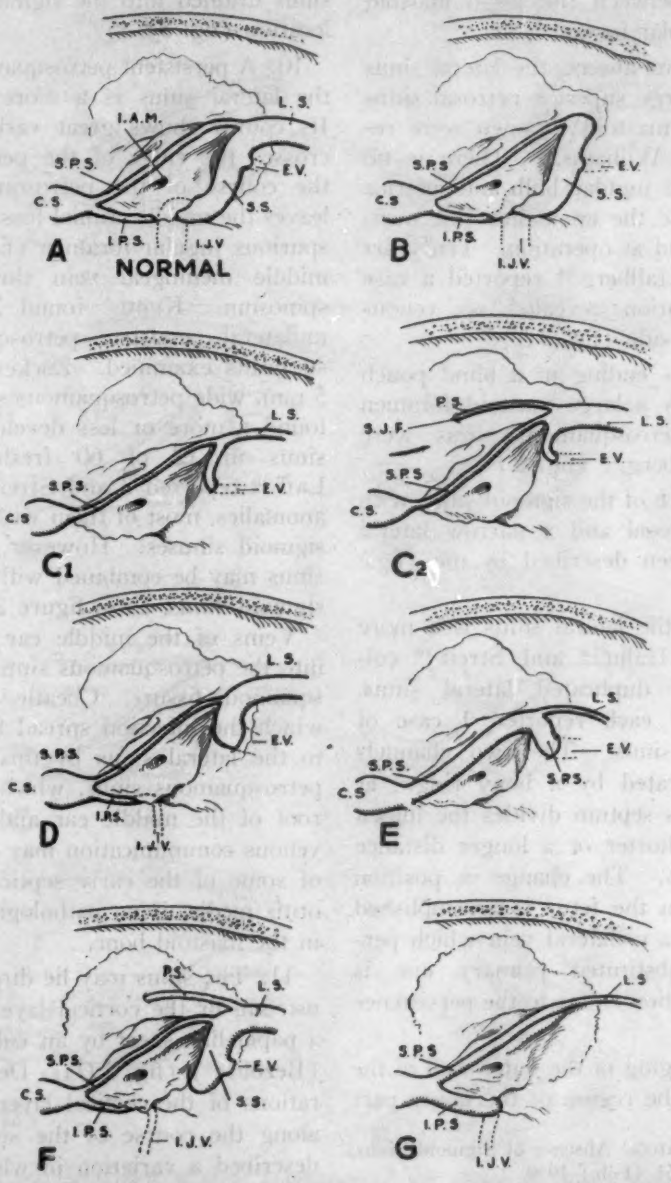

Fig. 2.—*A*, diagrammatic representation of the normal sinus region (intracranial view). Other drawings, m the same view, show anatomic variations of venous sinuses: *B*, lateral sinus absent. *C*[1], small transverse us leaving endocranium through mastoid foramen. *C*[2], persistent petrosquamous sinus present. *D*, petrous ne infantile. *E*, superior petrosal sinus passing through mastoid foramen. *F*, sigmoid sinus ending in a blind uch. *G*, complete absence of sigmoid sinus. The significance of the letters used in the drawings in figures 2 d 3 is as follows: *C.S.*, cavernous sinus; *E.V.*, mastoid emissary vein; *F.M.*, foramen magnum; *F.S.*, foramen nosum; *I.J.V.*, internal jugular vein; *I.P.S.*, inferior petrosal sinus; *L.S.*, lateral sinus; *P.S.*, petrosquamosal us; *S.J.F.*, spurious jugular foramen; *S.L.S.*, superior longitudinal sinus; *S.P.S.*, superior petrosal sinus; *S.S.*, gmoid sinus; *I.A.M.*, internal auditory meatus.

13. Hoople, G. D.: Ann. Otol., Rhin. & Laryng. **45**: 19, 1936.

14. Malacarne, M. V.: Encefalotomia nuova universale, Torino, G. Briolo, 1780; cited by Labbé.[2]

15 by 10 mm. In Laff's [15] case the lateral sinus abruptly curved downward into a large mastoid foramen; the jugular foramen was small, the jugular bulb was absent and the inferior petrosal sinus emptied into a small jugular vein. The whole petrous bone was infantile and underdeveloped, although the patient was 35 years old (fig. 2 *D*). In Barkow's [16] case the sigmoid sinus was missing between the large mastoid foramen and the jugular fossa.

5. The sigmoid sinus absent, the lateral sinus threadlike and the large superior petrosal sinus passing through the mastoid foramen were reported in a case of Williams.[17] There is no information about the jugular bulb and inferior petrosal sinus because the anomalies just mentioned were discovered at operation. Ten years later Williams and Hallberg [18] reported a case in which the operation revealed no venous sinuses on the right side (fig. 2 *E*).

6. A sigmoid sinus ending in a blind pouch and draining through a large mastoid foramen and a persistent petrosquamous sinus were reported by Furstenberg [12] (fig. 2 *F*).

7. Complete absence of the sigmoid sinus with a large inferior petrosal and a narrow lateral sinus has already been described by me (figs. 1 and 2 *G*).

8. Duplication of the lateral sinus is a more frequent anomaly. Hahn [19] and Streit [20] collected 12 cases of duplicated lateral sinus. Streit and Brown [21] each reported 1 case of duplicated sigmoid sinus. The two channels sometimes are separated by a bony ridge; at other times a fibrous septum divides the lumen of the sinus for a shorter or a longer distance (fig. 3 *A* [1] and *A* [2]). The change in position of the dural veins in the fetus is accomplished by the formation of a collateral vein which persists while the substituted primary one is obliterated. Duplication is due to the persistence of both these veins.

9. Hernia-like bulging of the outer wall of the sinus, especially in the region of the upper part of the knee, occurs with relative frequency (3 *B* [1]. Ruttin [22] described the following va tion: A huge sigmoid sinus ended in a b sac in the squamous portion of the temp bone. In addition, the jugular bulb was m ing and a small vein connected the enlar sigmoid sinus with the internal jugular v The superior petrosal sinus and the thin lat sinus drained into the sigmoid sac at the u location (fig. 3 *B* [2]).

10. A persistent petrosquamous sinus drain the lateral sinus is a more frequent anom Its course shows great variations. Usually crosses the ridge of the petrous bone, follo the course of the petrosquamous fissure a leaves the middle cranial fossa either through spurious jugular foramen (fig. 3 *C*) or with middle meningeal vein through the foram spinosum. Knott [5] found 7 bilateral and unilateral persistent petrosquamous sinuses 44 heads examined. Zuckerkandl [23] reporte 5 mm. wide petrosquamous sinus. Vernieuw found a more or less developed petrosquam sinus in 12 of 60 fresh heads examin Laff [15] reported 3 and Streit [20] reported 2 s anomalies, most of them with normal lateral a sigmoid sinuses. However, the petrosquam sinus may be combined with an anomaly of sigmoid sinus, as in figure 2 *F*.

Veins of the middle ear may drain direc into the petrosquamous sinus through the pet squamous fissure. Cheatle [7] reported 2 cases which the infection spread from the middle to the lateral sinus by this short route of petrosquamous sinus, which lies just above roof of the middle ear and the antrum. T venous communication may give the explanati of some of the early septic symptoms in act otitis media, if no pathologic condition is fou in the mastoid bone.

11. The sinus may lie directly under the pe osteum, or the cortical layer may be thinned a paper-like sheet by an enlarged sigmoid sin (Bezold [25]) (fig. 3 *D*). Dehiscences and per rations of the cortical layer are not uncomm along the course of the sinus. Furstenberg described a variation in which the lateral sin was located lateral to the skull in the soft tissu of the scalp.

The wide variations in size and location of t sigmoid sinus in the mastoid process are w known to the otologist. It may be, however, a certain interest to mention those anatomic var

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16. Barkow, J. C. L.: Anatomische Abhandlungen, Breslau, F. Hirt, 1851, p. 1.

17. Williams, H. L., Jr.: Apparent Unilateral Absence of Sigmoid Sinus Noted at Operation, Arch. Otolaryng. **12**:339 (Sept.) 1930.

18. Williams, H. L., and Hallberg, O. E.: Congenital Absence of Cranial Venous Sinuses on the Right, Arch. Otolaryng. **33**:78 (Jan.) 1941.

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20. Streit, H.: Arch. f. Ohrenh. **58**:85 and 161, 1903.

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23. Zuckerkandl: Monatschr. f. Ohrenh. **7**:102, 18

24. Vernieuwe: Rev. de laryng. **42**:207, 1921.

25. Bezold: Monatschr. f. Ohrenh. **7**:130, 1873.

of the other venous sinuses with which one ently deals during operations.

Absence of the superior petrosal sinus was ted in a few cases (Knott,[5] 2; Hyrtl,[4] 1); treit's[20] case the superior petrosal sinus d downward into the foramen spinosum of iddle fossa (fig. 3 *E*).

Theile.[26] Major variations of the inferior petrosal sinus are extremely rare.

4. Absence of the mastoid foramen and several foramens have been frequently reported. According to Coudert,[27] the mastoid foramen is relatively better developed in children than it is in adults. Occasionally the mastoid emissary vein

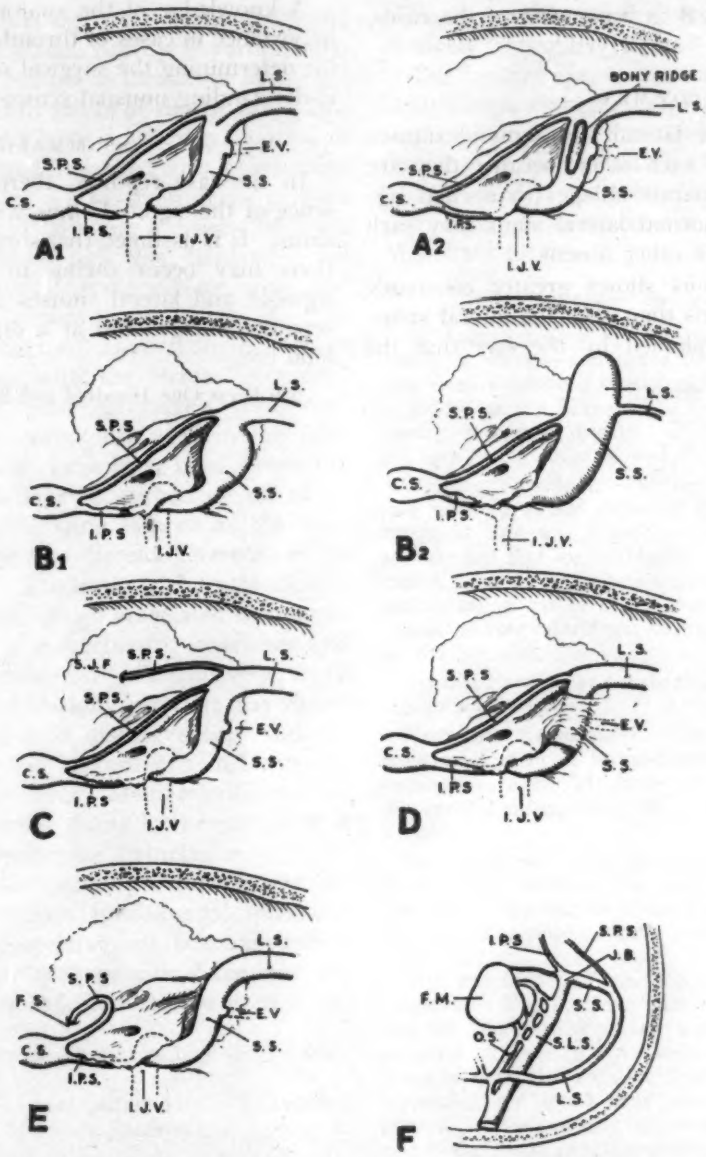

Fig. 3.—Diagrammatic representations of other anomalies of the region of the sinuses (intracranial view). Duplications of lateral and sigmoid sinuses. *B*, hernia-like bulging of outer wall: (*1*) in upper part of region ee and (*2*) with jugular bulb missing. *C*, persistent petrosquamous sinus draining lateral sinus. *D*, cortical thinned to paper-like sheet by enlarged sigmoid sinus. *E*, superior petrosal sinus turning downward into men spinosum of middle fossa. *F*, superior longitudinal sinus continuing directly with jugular bulb.

The superior longitudinal sinus continued ctly with the jugular bulb in a case of Streit 3 *E*).

The inferior petrosal sinus drained into the rior thyroid vein in a case reported by

crosses the mastoid cells in a long bony canal, and an isolated thrombophlebitis of the emissary vein may occur. In many cases the emissary

26. Theile, cited by Streit.[20]

27. Coudert, R.: Anatomie et pathologie de la veine émissaire mastoidienne, Thesis, Paris, no. 492, 1900.

vein was found lying on the dura of the posterior fossa for variable distances. Both the internal and external openings of the mastoid foramen show the widest variations in location. A direct connection between the emissary vein and the petrosquamous sinus was described by Otto.[28]

5. The ophthalmopetrosal sinus connects the ophthalmic vein with the superior petrosal or the lateral sinus in 8 to 9 per cent of the cases, according to Knott [5] and Hyrtl.[4]

COMMENT

Variations of the lateral and sigmoid sinuses are independent of each other, because they are developed from separate anlages; a normal sigmoid sinus or a normal lateral sinus may each be present with the other absent.

The sigmoid sinus shows greater constancy and fewer variations than does the lateral sinus. This could be explained by the fact that the lateral sinus has to adapt itself to the increa size and changing form of the surrounding s tures, for example, the brain and the otic cap Therefore, the lateral sinus is more likely t interfered with in its development than is sigmoid sinus. The latter is located close to base of the brain from the very beginning o development.

A knowledge of the anatomic variations importance in cases of thrombophlebitis, not for determining the surgical management bu understanding unusual symptoms and signs.

SUMMARY

In the case reported there was complete sence of the sigmoid sinus with a narrow la sinus. It is assumed that similar anatomic va tions may occur owing to the fact that sigmoid and lateral sinuses are developed separate anlages and at a different stage of fetal life.

620 West One Hundred and Sixty-Eighth Stree

28. Otto, cited by Streit.[20]

MÉNIÈRE'S DISEASE

HISTOPATHOLOGIC OBSERVATIONS

J. R. LINDSAY, M.D.

CHICAGO

eginning with the discovery by Hallpike and rns[1] of a labyrinthine dropsy (hydrops laby-hi) in the affected ear in 2 cases of Ménière's ase, a number of reports of similar histopath-ic observations[2] have appeared. A total of ars have now been described with labyrinthine psy of this type, 11 of them from persons ved to have had Ménière's disease, 4 from sons who probably had this disease[2c,d] but n insufficient history for complete proof and rom a person without any history.[2e] The n "Ménière's disease" as used in this report ot synonymous with the terms "Ménière's ptom complex" and "Ménière's syndrome" hey have been commonly used in the past. e latter two terms have been used frequently include labyrinthine disturbances of toxic, cular and other origins as well as the idio-hic type of vertigo herein referred to as nière's disease. The latter is characterized by urring attacks of vertigo associated with audi-y disturbances of a fluctuating character and now been shown fairly conclusively to be ed on hydrops of the labyrinth. It may there-e be considered as a disease entity, and the ne of Ménière has become attached through mon use. The term "Ménière's disease" is ein restricted to this entity to the exclusion of er types of labyrinthine disturbance.

Although the histopathologic reports are in-asing slowly, certain fundamental questions arding the disease have not been answered. e causation and pathogenesis have not yet n clearly determined, nor has the precise ex-planation for the clinical disturbances been conclusively indicated.

The further examination and recording of histopathologic changes in proved cases is one of the ways by which these deficiencies in the basic information may be filled.

This report presents the clinical and histopathologic observations in a proved case of Ménière's disease.

REPORT OF A CASE

In August 1942 a 47 year old man fell during an attack of vertigo, suffered a fracture of the skull and died from a subdural hematoma.[3] In 1939 he had been examined at the Mayo Clinic[4] and was considered as having Ménière's disease. Functional examination of the ears at that time showed the following:

Hearing Threshold.—The right ear was normal for low tones up to and including 2048 cycles. An abrupt threshold loss was noted for frequencies above 2048 and marked loss for all higher tones. The left ear revealed a loss of 30 decibels for low tones, including 2048 cycles, and a rather sharp loss for tones above 2048 cycles. A cold caloric test showed a diminished response on the left side.

General Physical and Neurologic Examinations.—The results were negative.

Histologic Examination.—The temporal bones were fixed in solution of formaldehyde and prepared in the usual way. The left bone was serially sectioned in the vertical plane, while the right was sectioned horizontally.

(*a*) Right ear: The eardrum and the outer wall of the tympanic cavity on the right were missing from the specimen. The mucosa of the middle ear was thickened, and the remaining air cells contained degenerated red blood cells.

The membranes of the inner ear were fairly well preserved. The cochlear duct, the saccule, the utricle and the semicircular canals were normal in size and contour. A few red blood cells were seen in the scala tympani near the cochlear aqueduct. Masses of extravasated red blood cells were seen in the internal meatus, in the vascular spaces of the lamina cribrosa and in the spiral ganglion of the upper basal and lower middle coils. The vessels in the modiolus were distended with blood. Corti's organ was well preserved. The sensory epithelium showed early postmortem degenerative changes. The spiral ganglion appeared normal in the upper portion, but the cells in the basal coil were greatly reduced in number (fig. 1 *A*).

Summary: The pathologic changes in the right ear were limited to the vascular congestion and hemorrhage

From the Division of Otolaryngology of the Uni-ity of Chicago.

1. Hallpike, C. S., and Cairns, H.: Observations the Pathology of Ménière's Syndrome, J. Laryng. & ol. **53**:625-634 (Oct.) 1938.

2. (*a*) Yamakawa: Ztschr. f. Oto-Rhino-Laryng. 181, 1938; cited by Rollin.[2c] (*b*) Hallpike, C. S., Wright, A. J.: Histologic Changes in Temporal nes of Case of Ménière's Disease, J. Laryng. & Otol. 59-66 (Jan.) 1940. (*c*) Rollin, H.: Zur Kenntnis Labyrinthhydrops und des durch ihn bedingten nière, Hals-, Nasen- u. Ohrenarzt (Teil 1) **31**:73- (March) 1940. (*d*) Lindsay, J. R.: Labyrinthine opsy and Ménière's Disease, Arch. Otolaryng. **35**: 857 (June) 1942. (*e*) Altmann, F., and Fowler, P., Jr.: Histological Findings in Ménière's Symptom mplex, Ann. Otol., Rhin. & Laryng. **52**:52-80 (March) 3.

3. The temporal bones were made available through Dr. R. L. Boies, of the University of Minnesota.

4. A report of the functional examination was supplied by Dr. H. L. Williams, of the Mayo Clinic.

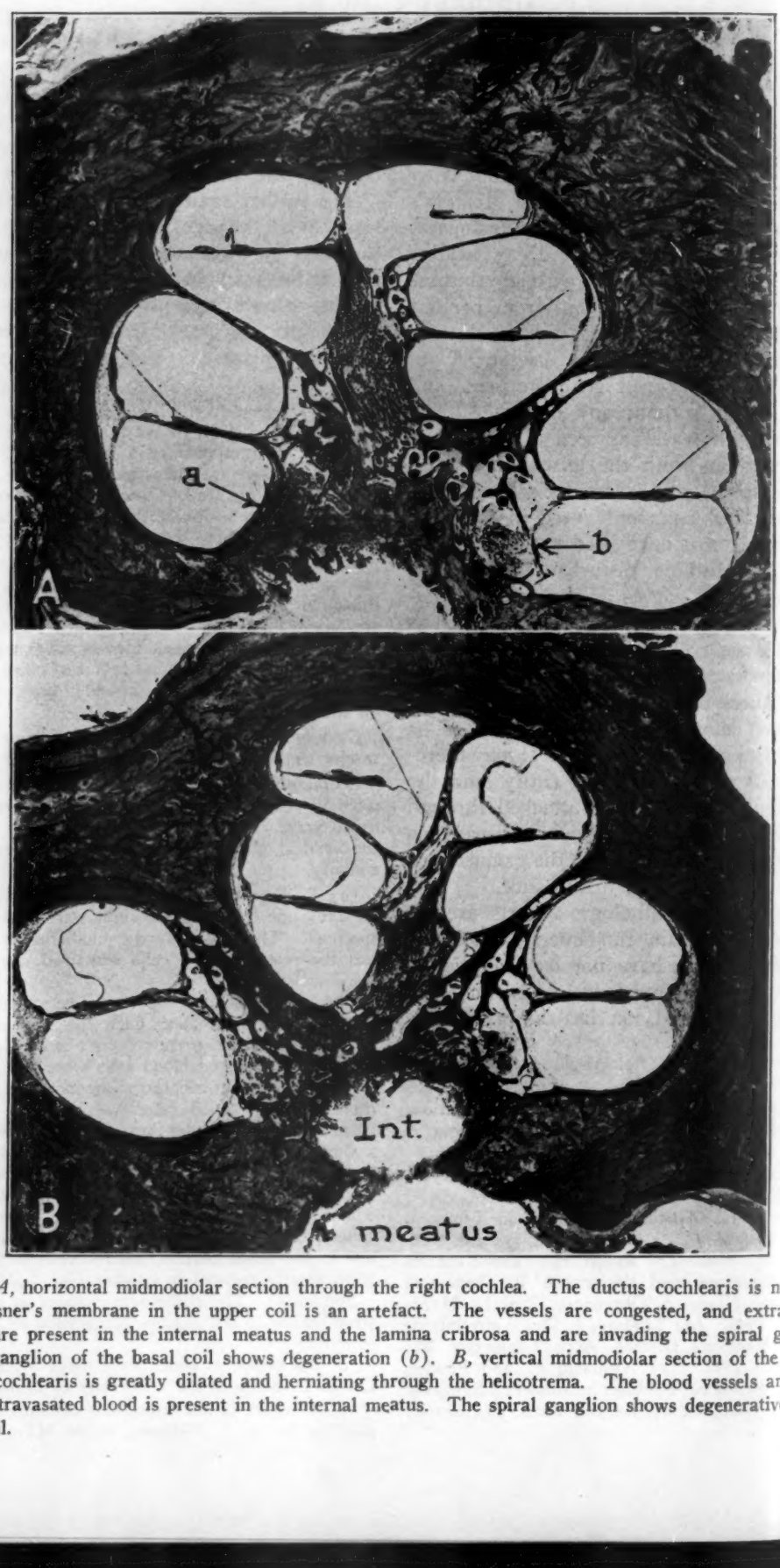

Fig. 1.—*A*, horizontal midmodiolar section through the right cochlea. The ductus cochlearis is normal. T loss of Reissner's membrane in the upper coil is an artefact. The vessels are congested, and extravasated blood cells are present in the internal meatus and the lamina cribrosa and are invading the spiral ganglion The spiral ganglion of the basal coil shows degeneration (*b*). *B*, vertical midmodiolar section of the left coch The ductus cochlearis is greatly dilated and herniating through the helicotrema. The blood vessels are congest and some extravasated blood is present in the internal meatus. The spiral ganglion shows degenerative changes the basal coil.

modiolus and to the degenerative changes in the ganglion. The former are explained by the ter- cranial injury, while the changes in the ganglion probably related directly to the hearing loss for above the frequency of 2048 cycles.

Left ear: The structures of the middle ear partly missing from the specimen. The mucosa ed some thickening, and there were small collec- of degenerated red blood cells.

membranes of the internal ear were well pre- d. The cochlear duct was greatly dilated and in upper basal coil had reduced the scala vestibuli to row channel. There was beginning herniation of ochlear duct through the helicotrema (fig. 1 *B*).

backward below and lateral to the utricle and herniated for a short distance into the small end of the horizontal semicircular canal (fig. 4). The utricle was dilated in the lower portion leading to the ampulla of the posterior canal, where a tendency to herniate behind the thick wall of the ampulla was seen (fig. 4). At the upper part of the utricle near its junction with the ampullae of the superior and horizontal canals the walls had given way, and herniations were seen (figs. 4 and 5) which tended to project behind the thick walls of the ampullae. The lining epithelium appeared to be intact in these areas.

The walls of the semicircular canals were normal.

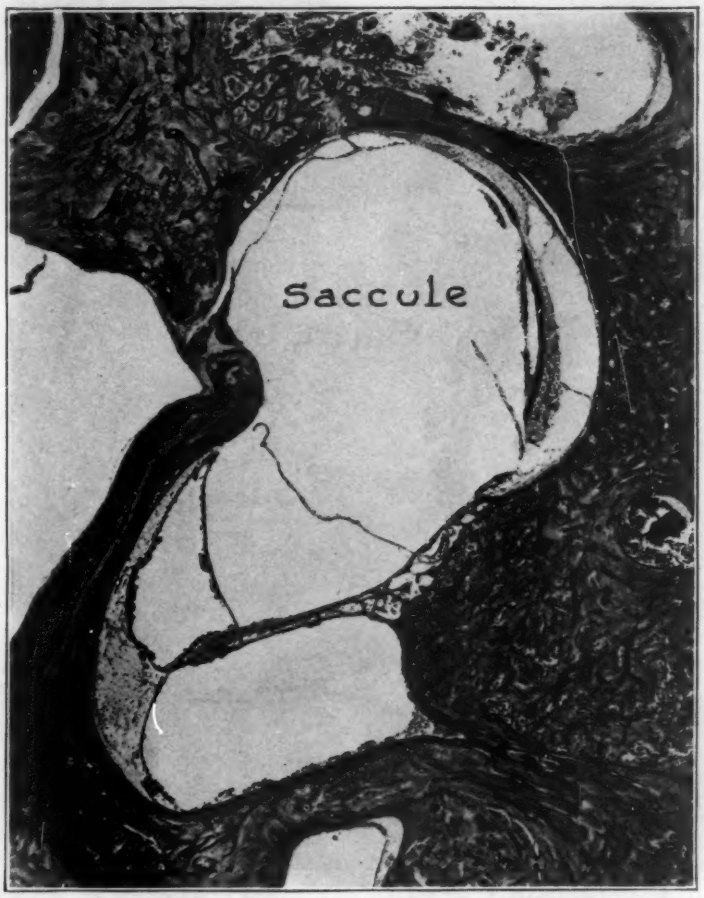

Fig. 2.—Section through the left vestibule showing the dilated saccule and the saccular macula.

orti's organ was present throughout, and the hair s seemed to be well preserved except for early post- tem degeneration. The blood vessels in the modiolus the stria vascularis were congested, and small col- ions of extravasated red cells were seen along the ls of the cochlear duct in the basal coil and in the cule, also in the internal meatus and along the facial ve. Only a few red cells were seen in the perilymph, ected near the cochlear aqueduct. The spiral gan- n appeared to have a normal content of nerve cells the upper coils, but in the basal coil the cells were uced in number, the remaining space in Rosenthal's al being filled with connective tissue.

The ductus reuniens was normal in size. The saccule ost completely filled the vestibule. The wall of the cule overlay the stapedial footplate. It extended

The sensory epithelium of the maculas and cristas showed mild postmortem degenerative changes.

The stria vascularis appeared essentially normal.

The aqueductus endolymphaticus showed fewer convolutions in the second portion than are usually present, while the saccus had been mostly torn away with the dura.

Summary: The pathologic changes in the inner ear were of three types: (1) dilatation of the condolymphatic spaces with resulting distortion of membranes, (2) degeneration of the spiral ganglion in the basal coil and (3) generalized vascular congestion and extravasation of red blood cells.

In vascular congestion and extravasation of red cells the left was comparable to the right ear, and these changes were due to the terminal cranial injury.

COMMENT

The histologic observations in the cochlea in this case correspond closely to those in the other cases which have been reported. The dilation of the ductus cochlearis had not progressed to the point of obliterating the scala vestibuli as it had in many of the reported cases. The case reported by Hallpike and Wright [2b] also showed a still lesser degree of dilatation of the ductus cochlearis. These cases precluded the possibility that the complete obliteration of the scala vestibuli is an essential factor in the disturbance of function. The hearing loss for low tones up to 2048 cycles in the affected ear must have been associated with the hydrops of the labyrinth. Such a loss for low tones has been recognized as a primary characteristic [5] of Ménière's disease. The loss for high tones was a bilateral finding. The degree of threshold loss cannot be closely correlated with the degree of hydrops since the hearing test had been made three years earlier. As is a characteristic [5] of this disease in early stages that the hearing threshold varies greatly, depending on the stage of an acute attack, a single test has always a limited value. It is necessary to have a number of tests, made at different stages in order to represent the threshold correctly. The fluctuation has been attributed to changes in tinnitus, but this is insufficient to explain more than a part of the variation which occurs. Such fluctuations in auditory threshold do not appear to be explainable on the basis of a lesion of the neuromechanism.

The loss for high tones in this case was a bilateral finding and therefore did not appear to have any relation to the hydrops of the labyrinth.[5a] It is not uncommon, however, to find that patients with unilateral Ménière's disease have a threshold loss for high tones in both ears

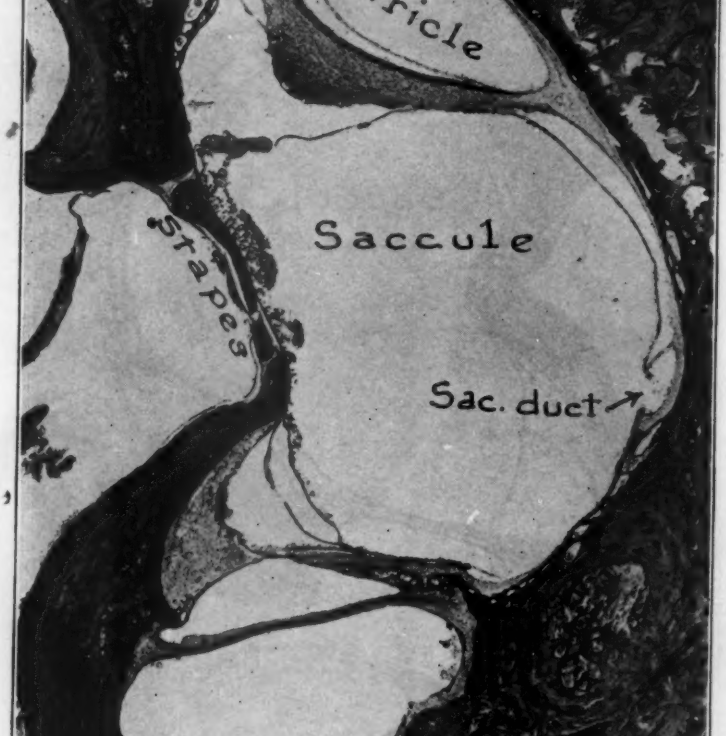

Fig. 3.—Section through the left vestibule slightly posterior to that in figure 2, showing the utricle and the dilated saccule. The perilymphatic cistern has been obliterated.

5. (*a*) Mygind, S. H., and Dederding, D.: Pathogenesis of Ménière's Disease and of Kindred Conditions in Ear and Rest of Body, Ann. Otol., Rhin. & Laryng. **47**:938-946 (Dec.) 1938. (*b*) Lindsay, J. R.: Ménière's Disease, Minnesota Med. **25**:778-783 (Oct.) 1942.

he bilateral degeneration of the spiral gan- in the basal coil in this case was undoubt- associated with the high tone deafness. he deafness of Ménière's disease has been ally described as of nerve type because of positive Rinne test. As was pointed out by ind and Dederding, however, the primary tone involvement and the fluctuations in shold do not seem compatible with a neural n. The fact that bone conduction is reduced g with air conduction indicates only that no inner ear. The histologic picture in this as well as in the two ears reported on earlier [2d] seems to lend support to this explanation of the auditory disturbances in that the neural elements appeared well preserved in contrast to the great distortion of the membranous structures. In place of the normal column of perilymph from the oval window to the round window, the column had been broken up or distorted by the dilated saccule and cochlear duct at the expense of the perilymphatic cistern and vestibular scala. Such an architec-

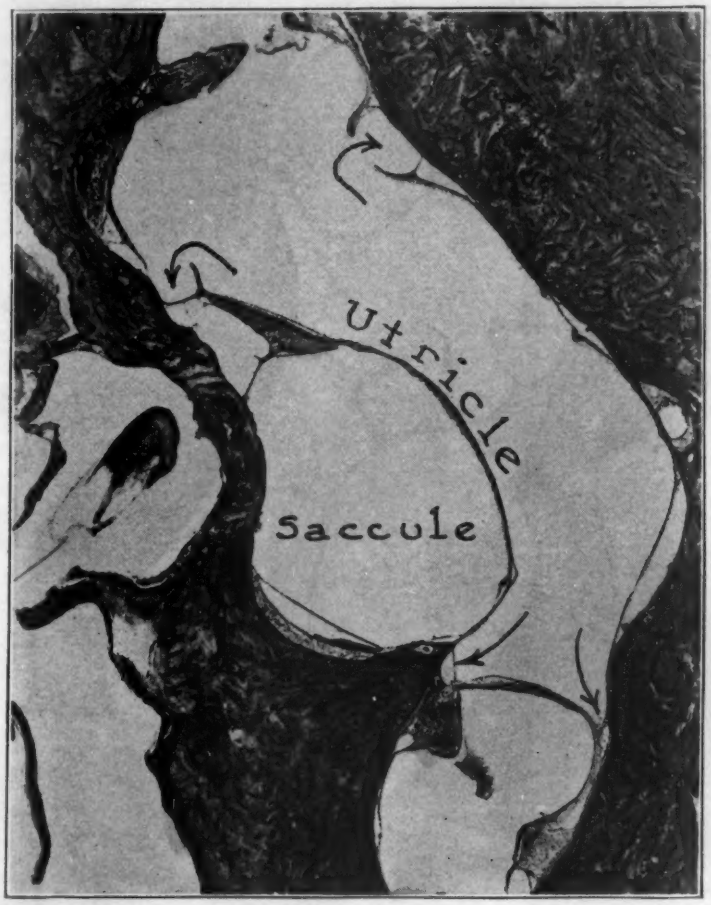

Fig. 4.—Section through the left vestibule slightly posterior to that in figure 3. The utricle shows herniation he ampulla of the posterior semicircular canal (lower arrows) and the ampulla of the superior and horizontal als (upper arrows).

turbance exists in the path of conduction as as the labyrinthine fluids. Comparison of ne and air conduction does not differentiate ween an impediment within the fluid medium the inner ear and a neural lesion. The posi- e Rinne test only localizes the lesion to the er ear or the perceptive apparatus in general l is the finding to be theoretically expected in e of some disturbance of conductivity in the id system. The clinical features of the deaf- ss appear more compatible with some such erference in the conduction medium in the tural rearrangement seems a probable basis for a disturbance in the function of the fluid column and thereby a loss of threshold.

The fluctuations in hearing are probably related to temporary variations in the amount of endolymph and therefore to variations in the degree of distortion of the membranous system.

The histologic changes found in the vestibular portion in the cases reported to date have shown greater variation. The dilatation of the saccule has been a fairly constant feature. The ductus reuniens and the saccular duct have been dilated

in some reported cases but not in others. The saccule has herniated into the small end of the horizontal semicircular canal in some, and in others there has been herniation of the utricle. The fact that some bones have been cut in the vertical and some in the horizontal plane makes comparison less easy.

In this case as in the 2 cases formerly reported by me there has been a consistent tendency for herniation to develop from the utricle adjacent to the ampullae of the semicircular canals with distortion of the outer wall of the ampullae. This distortion of the ampullary wall offers a reasonable explanation for the disturbances of vestibular function. Permanent distortion of the ampulla would explain impairment of the vestibular response in late stages while temporary distortion would result in an attack of vertigo and possibly also a temporary decrease in caloric response. The clinical observation that the attack of vertigo comes on during either an increase or a decrease of the deafness is compatible with this explanation, since it is during change in endolymph content in either direc that such a disturbance of the ampulla m occur.

The cause of the hydrops in this case is evident on histologic examination. The cochl aqueduct appeared to be patent, and the s vascularis showed no significant abnormali The endolymphatic aqueduct showed less p vascular connective tissue than is someti found, but in my previously reported cases aqueduct appeared entirely normal. It therefo appears doubtful if the pathogenesis can be e plained on such a basis.

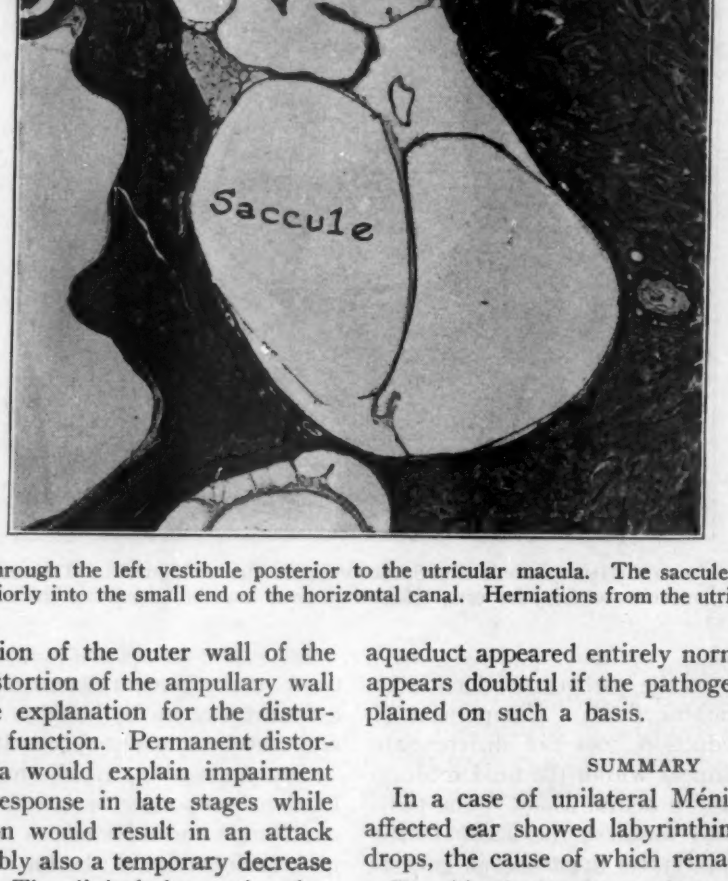

Fig. 5.—Section through the left vestibule posterior to the utricular macula. The saccule is still present a extends farther posteriorly into the small end of the horizontal canal. Herniations from the utricle are still prese

SUMMARY

In a case of unilateral Ménière's disease t affected ear showed labyrinthine dropsy or h drops, the cause of which remains unknown.

The histologic observations offer a probab explanation for the disturbances of function this disease.

INFECTION OF THE NOSE AND THROAT CAUSED BY PNEUMOCOCCI OF TYPE III

WILLIAM JEROME KNAUER, M.D.
JACKSONVILLE, FLA.

In April 1939 there came under my care a ient whose nose, pharynx, soft palate and gue were infected with pneumococci of type and whose condition was complicated by presence of pneumonia and nephritis. Six nths later I treated a second patient for a infection of the pharynx and the soft palate, ose condition was also complicated by pneu- nia. The unusual nature of the illnesses of se patients prompted me to record my obser- ions of the 2 cases, for during eighteen years practice I had seen nothing like them.

The average textbook of the ear, nose and oat and the textbooks of pathology merely ntion that nasal infections and infections of throat can be caused by pneumococci. No scription is given of the appearance of the oat or the nose, nor is treatment outlined. here is likewise a dearth of articles on this bject in medical periodicals. In the library one of the large medical schools of the country searched the literature in vain for mention of y detail of these infections.

In their textbook of bacteriology Zinsser and ayne-Jones [1] mentioned that the type III pneu- ococcus, Pneumococcus (Streptococcus) mu- sus, was first described by Howard and erkins [2] in 1901. This strain of pneumococcus as subsequently carefully studied by Schott- üller,[3] who isolated it in cases of parametritis, eritonitis, meningitis and phlebitis. It has nce been described by many authors as the citant of lobar pneumonia and a variety of her lesions, and often normally as an appar- tly harmless inhabitant of the mouth.

Although a tendency to occur in chains is haracteristic, morphologically micro-organisms of this type often appear on solid mediums in the diplococcus form. They are enclosed in an extensive capsule, which occurs with great regularity and persistence. Although they are not lance shaped, these bacterial organisms nevertheless greatly resemble the pneumococci. On solid mediums they have a tendency to grow in transparent moist masses. The regularity with which they ferment inulin makes it probable that it is more accurate to group them with the pneumococci than with the streptococci. Most of them share this inulin-fermenting characteristic with the pneumococci and are likewise soluble in bile. Occasionally a strain, such as the one studied by Dochez and Gillespie,[4] neither ferments inulin nor is soluble in bile. Rarely do these bacteria cause hemolysis. From the studies reported it must be concluded that this strain, distinguished from the pneumococci in the formation of a heavy mucoid colony and in the capsulation, is nevertheless more closely related to the pneumococci than to the true streptococci.

As would be expected from their capsulation, these organisms are highly virulent. Carter [5] in his textbook on microbiology observed:

> The pneumococcus is not a very hardy organism and has no natural existence outside of the animal body. In the finely divided spray thrown off from the nose and mouth, pneumococci live about one and one-half hours in the sunlight. In large masses of sputum they live for a month or more in the dark and about two weeks in the sunlight. They are very susceptible to ordinary germicides and are destroyed in ten minutes by a temperature of fifty-two centigrade. There seems to be a direct relation between capsule development and the resistance of pneumococci. This may explain why type III infections are accompanied by such a high mortality.

Type III pneumococci differ somewhat, then, from other pneumococci in that they are not lance shaped and are characterized by a wide capsule and by a slimy growth on culture mediums. In common with other pneumococci, however, they enter and leave the body by the same route, namely, through the mouth and the

Presented as a candidate's thesis to the American aryngological, Rhinological and Otological Society, c., 1942.

Presented at the meeting of the Southern Section of e American Laryngological, Rhinological and Oto- gical Society, Chattanooga, Tenn., Jan. 28, 1943.

1. Zinsser, H., and Bayne-Jones, S.: A Textbook of cteriology, New York, D. Appleton-Century Com- ny, Inc., 1939, pp. 319-320.

2. Howard, W. T., and Perkins, R. G.: J. M. Re- arch **6**:163, 1901; cited by Zinsser and Bayne-Jones.[1]

3. Schottmüller, H.: München. med. Wchnschr. **50**: 9, 1903; cited by Zinsser and Bayne-Jones.[1]

4. Dochez, A. R., and Gillespie, L. J.: A Biologic Classification of Pneumococci by Means of Immunity Reactions, J. A. M. A. **61**:727 (Sept. 6) 1913; cited by Zinsser and Bayne-Jones.[1]

5. Carter, C. F.: Microbiology and Pathology, ed. 2, St. Louis, C. V. Mosby Company, 1939, pp. 325-328.

nose. Practically every person becomes a carrier of pneumococci at some time during the year. Persons who have been in contact with a patient having pneumonia often carry the organisms in the throat for a few days or weeks. Most carriers who have not been in contact with such a patient harbor comparatively avirulent pneumococci and are of little danger, but pneumococci of type III may be present in their throats. That carriers of type III pneumococci are common while infections caused by pneumococci of type III are rare is a phenomenon difficult to explain. Usually the carriers harbor these organisms for a short time only. It was once believed that pneumonia was caused by autoinfection, that is, that the patient was infected by pneumococci harbored in his throat, but it is now known that autoinfection seldom occurs.

Recovery from a pneumococcic infection confers little if any immunity to the disease. The natural resistance of man to the pneumococcus is comparatively high, and a person probably never contracts a disease caused by this organism unless the resistance is lowered. Negroes are more susceptible than white persons to infection from this source, and men are more susceptible than women. The greater susceptibility probably results from greater exposure to the conditions that lower resistance. Delafield and Prudden [6] in their textbook of pathology stated that leukocytosis usually accompanies this type of infection and that it may induce a pseudomembranous inflammation of the mucous membranes.

Boyd [7] observed that in pneumococcic infection of the lung the cardinal factor pathologically is the outpouring of an inflammatory exudate; this process is progressive, and one part of the affected area may be in one stage while a more distant part may have reached another stage. The important objective is to learn to recognize how early or how late is the process, and this purpose is accomplished by determining the degree of freshness of the exudate.

For a long time there was a great difference of opinion as to the route by which the pneumococci reach the lung. Some authors held that there is first an infection of the blood and that the lung is attacked by the hematogenous route; others concluded that the bacteria reach the lung by way of the bronchial tree. The controversy was settled by the work of Blake and Cecil [8] in 1920, which was confirmed by the more re work of Robertson.[9] Performing their exp ments on monkeys, Blake and Cecil [8] inje carefully typed pneumococci through the wal the trachea and killed animals each day up the time of crisis and after that time in orde study the process of resolution in pneumo They reported that the pneumococci penetr the walls of the larger bronchi, enter the ly phatics of the interstitial framework of the l and then penetrate the alveoli.

In their textbook of pathology Smith Gault [10] dismissed the subject of coryza a acute inflammation of the nose and throat w the statement that there is usually a mixed flo in which Micrococcus catarrhalis, the staphy coccus and the streptococcus predominate. L frequently, they noted, the pneumococcus a Haemophilus influenzae are recovered. Th observed that the inflammatory process, togeth with all other acute inflammatory lesions, m be classified either as a simple catarrhal proc or as an ulcerative or a membranous type inflammation if the lesion is sufficiently sev to warrant this designation.

A report of the 2 cases that came under t observation follows:

REPORT OF CASES

Case 1.—Mr. D. B., aged 47, became suddenly on the night of April 10, 1939 and complained of severe chill, lacrimation, obstruction to nasal breathi and a severe sore throat. The temperature was 103 the pulse rate 110 and the respiratory rate 24. had had pneumonia in 1919.

Examination the next day revealed acute conjun tivitis with much lacrimation. Both nares were fil with a pale white exudative pseudomembranous form tion covering the turbinate bones and the septum a extending backward in the nasopharynx onto t pharynx, the tonsillar fossae, the soft palate and t uvula. This odd white pseudomembrane, in appearan unlike the membrane observed in Vincent's angi diphtheria and streptococcic infection, had no od but bleeding followed its removal.

Urinalysis disclosed the presence of albumin (2 plus Examination of the blood revealed a white cell cou of 16,500 with 98 per cent polymorphonuclear leukocyt

6. Delafield, F., and Prudden, T. M.: Text-Book of Pathology, New York, William Wood & Company, 1919, p. 249.

7. Boyd, W.: The Pathology of Internal Diseases, ed. 3, Philadelphia, Lea & Febiger, 1940, pp. 140-141.

8. Blake, F. G., and Cecil, R. L.: Studies on Experimental Pneumonia: (*a*) Production of Pneumococcus Lobar Pneumonia in Monkeys, J. Exp Med. **31**:445 (April) 1920; (*b*) Pathology and Path genesis of Pneumococcus Lobar Pneumonia in Monkey ibid. **31**:445 (April) 1920; (*c*) Production in Monke of an Acute Respiratory Disease Resembling Influen by Inoculation with Bacillus Influenza, ibid. **32**:6 (Dec.) 1920; (*d*) Pathology of Experimental Influen and of Bacillus Influenzae Pneumonia, ibid. **32**:7 (Dec.) 1920; cited by Boyd,[7] p. 140.

9. Robertson, O. H.: Recent Studies on Exper mental Lobar Pneumonia: Pathogenesis, Recovery an Immunity, J. A. M. A. **111**:1432-1437 (Oct. 15) 193 cited by Boyd,[7] p. 141.

10. Smith, L. W., and Gault, E. S.: Essentia of Pathology, New York, D. Appleton-Century Com pany, Inc., 1938, p. 445.

ecimens for culture were taken from the nose, asopharynx and the pharynx. From this material cultures of type III pneumococci were grown. e patient's temperature remained stationary at an ted level of 103 F., and his condition grew more despite the repeated administration of acetyl- ylic acid in doses of 10 grains (0.6 Gm.). The rate continued to be 110, and the rate of respira- was increased to 26 per minute. The patient was efore hospitalized, and an internist was called in ultation.

treatment two antiseptic gargles were alternated, consisting of sodium perborate and the other of Gm. of phenol and a sufficient quantity of a solu- resembling alkaline aromatic solution N.F. to make cc. The throat was swabbed daily with a 2 per solution of silver nitrate, and a 2 per cent solution nerbromin N.F. was ordered as a spray to be used t and morning. A medicinal ointment was used in nose. Sulfapyridine was given in doses of 1 Gm. tablets) every four hours for three doses and then Gm. every four hours thereafter. Ten grains of ium bicarbonate was given with each dose of sulfa- idine, and ½ grain (1 mg.) of phenobarbital was given every four hours. Fluids were forced to amount of 3,000 cc. daily, and the intake and the put were carefully charted.

Daily examination of the blood included red cell, white and differential counts and an estimation of hemo- bin. Determination of sulfapyridine in the blood and urine was made daily. Blood cultures were taken later were reported negative.

With this medication the patient improved greatly, on April 16 the pseudomembrane in the nose and throat had practically disappeared. He could athe through the nose, and the throat was no longer e. The administration of sulfapyridine was discon- ued. Eighteen hours later the patient began to plain of a sore tongue. Examination revealed a udomembrane extending around the gums and the er surface of the tongue. From specimens obtained m the gums and the tongue pure cultures of type pneumococci were grown. Treatment with 0.5 Gm. sulfapyridine and 10 grains (0.6 Gm.) of sodium rbonate every four hours was immediately resumed. 2 per cent solution of silver nitrate was applied to gums and the tongue 3 times a day.

On April 18 the membrane had extended over the ire tongue, which looked as if it were going to come one massive slough. The sinuses appeared dark transillumination and were painful, but no operative ocedure was attempted, as I presumed that they were ed with the same pseudomembranous exudate that s on the gums and on the tongue. Innumerable sts formed daily in the nose and had to be removed th forceps.

The patient gradually improved, and on April 21 the mperature was normal. By the following day all of e pseudomembrane had disappeared from around the ms, from underneath the tongue and from the greater rt of the tongue, except the tip. On April 23 actically none of the membrane remained, the tem- rature was normal and the patient was allowed to up. Vitamin B complex was given, and on the rning of April 24 treatment with sulfapyridine was scontinued.

After supper on that night the patient's temperature ddenly rose to 103 F., the pseudomembrane was rming on new areas of the tongue, the pulse rate as increased and the rate of respiration was slightly celerated. The patient complained of pain in the ft side of the chest. A blood culture was negative. Treatment with 0.5 Gm. of sulfapyridine and 10 grains of sodium bicarbonate was again resumed, a 2 per cent solution of silver nitrate was applied to the tongue and an antiseptic gargle was ordered. Examination of the lungs by the internist indicated congestion at the base of the left lung with consolidation about 2 inches (5 cm.) above the diaphragm. His report was confirmed by roentgen studies on April 25. Codeine and acetylsalicylic acid were given for the pain. The larynx was not involved.

Throughout the course of the disease the patient suffered severely from nausea caused by the sulfapyridine, and several reduced doses had to be resorted to on occasion before the regular dose could be resumed. Toward the end of the period of hospitalization he complained of nausea in milder form immediately following the use of the gargles and the spray. A gargle of sodium perborate in the strength of a teaspoon to a glass of warm water seemed to give relief and not to be nauseating.

Swabbing the throat once a day with a 2 per cent solution of silver nitrate seemed to relieve the condition in this area more than did any other therapeutic measure. This solution applied 3 times a day was the only treatment that helped to clear the tongue.

Although he was able to talk coherently, the patient was unaware of what went on around him from the second day of the illness until four days before dismissal from the hospital. Afterward, he had no recollection of his suffering. From the onset he was in an extremely toxic condition, and the least disturbance upset him greatly, causing an increased pulse rate, pallor and a state almost of shock.

On April 27 the temperature again became normal. With the continued improvement of the patient, all medication was discontinued two days later, and on May 1 he was allowed to go to his home. He was kept in bed at home for two weeks and thereafter experienced an uneventful convalescence.

CASE 2.—Mr. R. T., aged 84, was hospitalized on Oct. 24, 1939 for a prostatectomy, which was performed on Oct. 30. Convalescence was without untoward incident until November 7, at which time the patient had been up in a chair for two days and was making excellent progress. On that day he had a chill and complained of a sore throat. The temperature rose to 103.5 F.

Examination of the throat revealed a pale white pseudomembrane extending from each side of the uvula along the soft palate to both pillars and to the pharynx. The appearance of the throat almost duplicated that of the throat described in case 1. There was no evidence of pathologic change in the ears and the nose, and examination of the heart and the lungs gave negative results.

A pure culture of type III pneumococci was grown. Examination of the blood revealed a white cell count of 25,000 with 94 per cent neutrophils.

An antiseptic gargle composed of 0.52 Gm. of phenol and a solution resembling alkaline aromatic solution N. F. in sufficient quantity to make 360 cc. was prescribed, and a 2 per cent solution of merbromin N. F. was ordered as a spray to be used night and morning. An initial dose of 40 grains (2.4 Gm.) of sulfapyridine with 40 grains (2.4 Gm.) of sodium bicarbonate was given orally, and these drugs were then continued in doses of 10 grains (0.6 Gm.) each every four hours. Swabbing once a day with a 2 per cent solution of silver nitrate seemed to alleviate the condition of the throat more than did any other measure. Under this treatment the peculiar pseudomembrane gradually disappeared and there was less elevation of temperature.

At times extreme nausea followed the administration of the sulfapyridine and it was necessary to reduce the dose temporarily. Late in the course of the infection the patient experienced nausea immediately after using the gargles and the spray. He was able to tolerate a gargle of sodium perborate in the strength of a teaspoon to a glass of warm water and appeared to be relieved by this medication.

Extreme toxicity was present from the onset of the infection. The slightest disturbance caused the patient to become greatly upset, to exhibit pallor, to have an increased pulse rate and to enter a state bordering on shock. Although he was coherent in speech, he had no recollection of what went on around him while the infection was at its height in the throat, and later he did not recall his suffering.

On November 12, as the throat became practically clear of the pseudomembrane, bronchopneumonia developed on the left side. There was no involvement of the larynx. As the pneumonia progressed, the throat cleared, and on November 19 the patient was dismissed so far as attention to the throat was concerned. Suffice it to say that despite his age, of 84 years, he survived the pneumococcic infection of the throat and the bronchopneumonia as well. On December 7 he was discharged from the hospital.

COMMENT

Does the condition of the upper part of the respiratory tract described in these 2 cases occur rarely? With the improved technic of today, should more such cases be recognized? In the year and nine months since the second patient came under my care I have repeatedly and carefully examined the throats of patients with pneumonia, but I have failed to find a single person with the peculiar pale white exudative pseudomembrane extending from the nose or the soft palate to the pharynx with cultures producing a pure growth of pneumococci of type III.

This type of infection caused by the type III pneumococcus, with the pseudomembranous formation extending from the nose to the pharynx and the tonsillar fossae and to the tongue, accompanied by extreme toxicity of the patient, as described in case 1, was a condition I had not observed before. Finding no definite description in textbooks nor other help from the literature, I was without guidance in treating the patient in this case. In my opinion he would have died without treatment with sulfapyridine, as would also the patient in case 2. Had it been possible to examine the inside of the sinuses, I believe I should have found them lined with a pseudomembrane similar to that of the nose and throat. The condition of the throat as it appeared in both of these cases suggested no condition observed previously in my experience except that it somewhat resembled that of agranulocytic angina. The evidence of infection on the tongue of the first patient was the most striking that I had ever witnessed.

Are some of the fatal cases diagnosed as instances of diphtheria in which there is a membranous condition of the throat with cultu failing to reveal the organisms for this dise actually cases of infection caused by the type pneumococcus? Perhaps, if a bacteriologist w to make a thorough examination the illness co be ascribed to this micro-organism. At a gla one might suspect the pseudomembrane to that of agranulocytosis or of Vincent's angi but the severely ill patient, the high temperat and the high leukocyte count enable one to r out the possibility of the former condition, a in the latter disease this syndrome is ra observed. Leukocytosis was present in both the cases herein described. The patient w diphtheria is usually not as ill at first as w the patients in these 2 cases, and as a rule t temperature is not as high. Also, a strep coccic infection of the throat is generally n characterized by an extensive membrane, a the patient's chief complaint is an extremely s throat. Toxicity, however, accompanies bo pneumococcic and streptococcic infections.

Could the complication of pneumonia ha been prevented in these cases? If so, the qu tion arises as to what measures would have be most efficacious as a means of prevention.

SUMMARY

Lesions of the nose and throat characteriz by an exudative pseudomembrane and caus by pneumococci of type III appear to be ra The condition is barely mentioned in textbook

The onset of the disease is sudden, and t patient's condition becomes extremely tox immediately.

The exudative pseudomembrane resembl somewhat the membrane observed in agranul cytic angina, and the surface from which it removed bleeds easily.

Specimens for culture should be obtained once from this type of lesion, and a compete bacteriologist should examine the cultures.

The administration of sulfapyridine is appa ently of great help in the treatment of an infe tion of this nature.

Antiseptic gargles and the application of mild solution of silver nitrate seem to provid helpful local treatment.

The patient with this disease should be imm diately hospitalized and isolated.

Careful nursing and attention to the minute detail are essential in saving the life of th patient.

The services of a skilled internist are require throughout the course of the disease for th general treatment and the welfare of the patien

Buckman Building.

TONSILLECTOMY AND POLIOMYELITIS

ONE CASE OF POLIOMYELITIS FOLLOWING 8,915 TONSILLECTOMIES *

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NEW YORK

The occurrence of poliomyelitis, in its worst
n, within a few weeks after tonsillectomy in
amily of 6 children, 5 of whom had been
rated on for the removal of their tonsils and
noids has been strikingly reported by Krill
Toomey.[1] Bulbar poliomyelitis developed
ll of the 5. Two died on the seventeenth day
r the operation and 1 on the eighteenth day.
parents and the sixth, and youngest, child,
years old, who had not been operated on,
aped the disease.

V. L. Aycock [2] has reviewed and analyzed
ilable data in relation to the incidence of
iomyelitis following tonsillectomy, and Sey-
,[3] with the many such instances of the disease
rred to in his article, has added to the
ousness of the discussion, as has an editorial [4]
The Journal of the American Medical Asso-
ion.

That additional information might be gained
a subject of such importance, the records of
Manhattan Eye, Ear and Throat Hospital
e been consulted, and many thousands of
ients on whom tonsillectomy had been per-
med have been communicated with by mail.
From the Department of Health of the city of
w York the number of cases of poliomyelitis
t occurred in the five years from 1937 to
1 inclusive was obtained from Dr. Green-
g, Epidemiologist of the Bureau of Prevent-
Diseases. He regretted that he could not
the proportion of cases of fatal bulbar
iomyelitis, the data having not been analyzed
that regard. His report follows:

Year	Cases	Deaths
1937	243	21
1938	43	4
1939	184	18
1940	67	5
1941	404	8

An increase in the number of cases every other year is conspicuously shown in this report. As 1937, 1939 and 1941 were the years in which the disease was most prevalent, the patients who had tonsillectomies performed on them during those years were communicated with by mail; 27,849 cards were sent out by the Manhattan Eye, Ear and Throat Hospital, requesting that answers to questions be written on a return postal card. On the return card were the patient's name, his age, his hospital number, the date of the tonsillectomy and the following questions. Have you had any illness since the tonsillectomy? What were the nature and the date of illnesses, if any?

A total of 8,915 replies were obtained, and among them 1 instance of poliomyelitis was reported, with the following data:

Age of patient, 7 years; date of tonsillectomy, July 27, 1937; illness, "infantile paralysis," late in August 1937.

A further inquiry was made to find out the extent of the paralysis and the present condition of the child, but no reply was obtained.

For the same year (1937), in which 243 cases of poliomyelitis with 21 deaths were recorded by the New York Department of Health, 10,597 cards were sent out and 2,545 replies were received. The replies reporting other illnesses in 1937, in addition to the 1 instance of poliomyelitis, gave the following data:

Report on Later Illnesses

Age of Patient, Years	Date of Tonsillectomy
6	Aug 16, 1937—"Spinal meningitis, December 1941"
3	April 7, 1937—"Spinal meningitis, double mastoid and whooping cough, 1938; measles, 1941; appendectomy, 1942"
6	Nov. 23, 1937—"Scarlet fever (?), measles, February 1938; double mastoids and spinal meningitis, March 1938; another mastoid, January 1939"
6	April 22, 1937—"Abscess in ears cleared up; other ailments due to spinal condition which causes nervousness remain"
21	June 19, 1937—"Deceased Feb. 17, 1938 from tubercular meningitis"
7	March 26, 1937—"April 26, 1940 to May 14, 1940 mastoids on left side, which ran into meningitis; disease almost fatal"

* From the records of the Manhattan Eye, Ear and oat Hospital.

1. Krill, C. E., and Toomey, J. A.: Multiple Cases Tonsillectomy and Poliomyelitis, J. A. M. A. **117:** (Sept. 20) 1941.

2. Aycock, W. L.: Tonsillectomy and Poliomyelitis, ficine **21**:65-94 (Feb.) 1942.

3. Seydell, E. M.: Relation of Tonsillectomy to omyelitis, Arch. Otolaryng. **35**:91-106 (Jan.) 1942.

4. Tonsillectomy and Poliomyelitis, editorial, J. A. A. **118**:980-981 (March 21) 1942.

For 1939, the year in which 184 cases of poliomyelitis with 18 deaths were reported by the New York Department of Health, 9,752 cards were sent out and 2,870 replies were received. Two persons made the following reports:

Age of Patient, Years	Report on Later Illnesses Date of Tonsillectomy	
5	Sept. 1, 1939—	"Whooping cough, 1940; chickenpox, 1941; pm. [sic] meningitis, Feb. 23, 1943"
5	Oct. 10, 1939—	"Gland infection and spinal meningitis, May 1940"

For 1941, when 404 cases of poliomyelitis with 8 deaths were reported by the New York Department of Health, 7,500 cards were sent out, and 3,500 replies were returned, with no illne reported.

The data in the 7 replies that reported illne after tonsillectomies are summarized as follo All mentioned some spinal involvement. reported spinal meningitis, the earliest insta occurring with "double mastoids" three mo after the tonsillectomy and the latest over years after the tonsillectomy. One repo "tubercular meningitis" in a man aged 21, died eight months after the tonsillectomy. reported "a spinal condition which causes vousness" in a child aged 6. One repo "infantile paralysis," which occurred in a aged 7 about a month after his tonsils and noids had been removed.

127 East Sixty-Second Street.

A NEW METHOD FOR THE REPAIR OF A SMALL LOSS OF ALAR RIM

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The method presented here is useful only for repair of a very small loss of alar rim, a perhaps no more than a quarter of an inch

requires a complicated or an extensive surgical operation. After many procedures had been experimented with, the method described in the

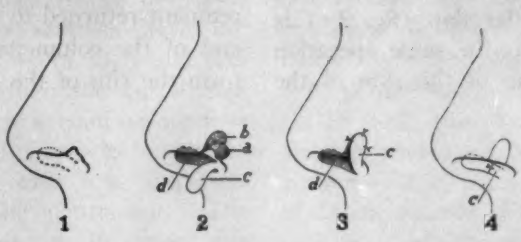

Fig. 1.—First stage. This shows the small loss of alar rim. The broken lines at the side of the columella ne the columella flap that is to be used.

Fig. 2.—First stage continued. At the site of the defect in the rim a small hinge flap (*a*) is turned down, ng the raw donor area (*b*). The columellar flap is indicated as *c* and the donor area for this flap as *d*.

Fig. 3.—First stage concluded. The columellar flap (*c*) has been migrated to cover the area of defect, while hinge flap (*a*) serves as a lining for the raw surface of the columellar flap (*c*). The donor area (*d*) of columellar flap is covered with an epidermic cutaneous graft to avoid leaving a raw area.

Fig. 4.—Second stage. The pedicle of the columellar flap (*c*) has been severed and returned, while the ainder of this flap is folded within the nostril to form the free margin of the rim and its lining.

Fig. 5.—A preoperative view of a loss of the left alar rim.

Fig. 6.—The defect just prior to the second operation. The pedicle of the columellar flap may be seen extend- from the alar rim to the columella.

Fig. 7.—The patient several weeks after the second stage. The remnant of the pedicle has been returned the flap folded in to form the rim.

width and a half-inch in height. Such a minor of alar rim presents a troublesome problem that the repair of so slight a defect often

following paragraph was finally evolved. The technic is simple and is easily carried out with little or no discomfort or inconvenience to the

patient. It is practically an office procedure. Furthermore, no additional disfigurement is produced even if the operation should result in total failure.

Briefly, the method utilizes a small flap of skin taken from the side of the columella, with the pedicle toward the base of the columella (fig. 1). It will be recognized at once that the procedure is limited by the size of the flap which one may obtain from the skin of the columella without encroaching on either the mucous membrane of the nose or the lower free margin of the columella. Two simple stages are required. At the first stage the columellar flap (fig. 2 *c*) is outlined and raised up. At the same operation a hinge flap (*a*) consisting of the skin of the outer surface of the nostril is turned down; hinge or pedicle of this flap is at the free mar of the defect. The columellar flap (*c*) migrated to the donor area (*b*) of the hinge which has been turned down and now lines columellar flap (fig. 3). At this time a small dermic free graft can be used to cover the do area (*d*) of the columellar flap. Petrola gauze packing is used to support the pedicle the free graft.

After an interval of about two weeks, pedicle of the columellar flap is severed and remnant returned to the donor area. The o end of the columellar flap is rolled inward form the rim of the nostril (fig. 4).

THE INDUSTRIAL NOISE HAZARD

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ndustrial management became vitally inter- d in safety engineering only after the coinci- ce of a low accident rate with increased output recognized. Just so, exposure to intense e is an industrial problem being by-passed management because executives have not rly realized the possibility of increased effi- cy and production through the curtailment oise. It is evident that in certain occupations ciated with continual exposure to loud noise eafness results which with long exposure progressive and may be permanent. The ker's deafness, because of its innocuous in in the little used high frequency range, s not stimulate executive interest as does a porary injury, such as a broken arm, with ch the relationship between cause and effect ore evident. Increased production, accentu- g the use of tools such as the pneumatically rated hammer, the chipper and the riveting in confined areas, underlines the importance he noise hazard in industry.

FACTS AND FINDINGS

From a historical standpoint the relationship ween exposure to intense noise and loss of ring has been known for at least one hundred rs. From 1882 to 1915 the preponderance tests were conducted on boiler makers, and consistent finding of impaired hearing led the coinage of the term "boiler maker's deaf- s." The first World War raised interest in fness caused by exposure to noise to a new ch. In the field of production it was found t factory machinists, locomotive engineers, ctor drivers, riveters and steel workers were fering a more or less typical loss of hearing. cognition of the fact that the guns of war sed aural trouble led to a comparison of the sults of exposure to the noise of firearms in il life and in combat. Sportsmen, policemen d other persons using firearms showed some ficiency in hearing, and a loss of hearing from suspected sources was also detected.

Recently facts have been accumulated which p to explain the phenomenon of occupational fness. A threshold of painful-feeling sound been established, varying from 90 to 135 decibels, depending on the frequency.[1] At the same time, it should be noted that the threshold of fatigue, though subject to considerable individual variation, is on the average 20 decibels below the threshold of feeling.[2] Therefore, the sensation of pain from noise is an unreliable index for the evaluation of aural protection.

Experimentation with animals exposed to noise[3] and the work of Crowe, Guild and Polvogt revealed actual destruction and degeneration of the organ of Corti in the basal coil of the cochlea. In correlation with pitch, acoustic trauma is characterized by a curve which is defective for high tones, usually beginning at a frequency of 1024 cycles per second, running a spiked course and reaching a peak of depression at 4096. This observation is verified by studies of controlled exposure to sound.[4] There are several factors concerned in this condition. The first, and probably the most prominent, factor is the effect of the intra-aural muscles, the stapedius and the tensor tympani. Hallowell Davis stated:

> The net effect of the contraction of the intra-aural muscles can best be described as a shift of the frequency toward the high tone end of the scale. The acoustic reflex apparently serves to protect the inner ear from damage from excessive amplitude of vibration which might be caused by intense tones of low pitch.[5]

The curves of acoustic trauma indicate that the effectiveness of this mechanism is limited to tones below 1024 cycles per second. Another factor to be considered is the anatomic condition, offering greater exposure of the nerve endings at the cochlear base, where sound waves enter. Also, there is a vascular bifurcation at this area, which, associated with a narrowing of the cochlear duct

1. Wegel, R. L.: The Physical Examination of Hearing and Binaural Aids for the Deaf, Proc. Nat. Acad. Sc. **8**:155, 1922.

2. Ewing, A. W. G., and Littler, T. S.: Auditory Fatigue and Adaptation, Brit. J. Psychol. **25**:284, 1935.

3. Horton, G. P.: An Experimental Study of Stimulation Deafness in Guinea Pigs, Ann. Otol., Rhin. & Laryng. **44**:252, 1935.

4. Perlman, H. B.: Acoustic Trauma in Man: Clinical and Experimental Studies, Arch. Otolaryng. **34**:429 (Sept.) 1941.

5. Stevens, S. S., and Davis, H.: Hearing: Its Psychology and Physiology, New York, John Wiley & Sons, Inc., 1938.

at this point, has been brought out as a possible predisposing weakness.[6]

NOISE SURVEY

A systematic estimation of levels of noise was obtained by means of the sound level meter of General Radio, Type 759. The intensity of the noise was recorded for trades in which noise was considered to be at a high level by the safety engineers. Special attention was given to certain occupations, as will be brought out later. To evaluate the findings correctly, chart 1 was drawn on a comparative basis with the conversational voice, at a distance of 10 feet (3 meters) used as a commonly known level of sound.

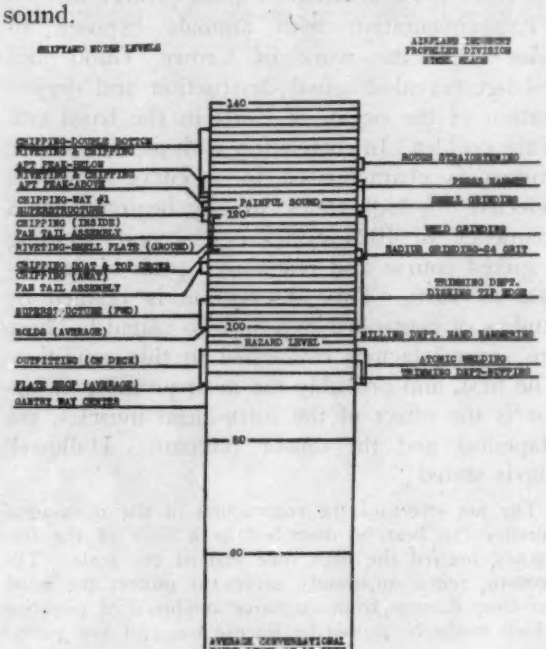


Chart 1.—Hearing in relation to noise.

In the shipyards, where the metal worked on was in the form of a large curved shell resting on separated points, the soundbox effect created beneath, where other men were working, was sufficient to raise the level of noise above that produced by work on a similar flat plate. The level of noise below a plate was greater than that above it. The intensity of the noise varies with the number of workers in an area, but two chippers working in the same space do not produce double the intensity of noise produced by one. This is characteristic of the logarithmic nature of the intensification of sound. Two operations with the same level of noise running simultaneously give an additive effect of approximately 3 to 4 decibels above that of one alo Fewer intervals free from noise were afford however. Confined areas, such as dou bottoms, deck houses, fore peak and assemblies, prevented rapid escape and dispers of the noise and hence produced the high levels of noise.

The chipping hammer heads the list of no producers in the shipyard. One hammer p duces from 110 to 135 decibels of sound, depe ing on the material being worked and the po of the hammer. The man using the hamm usually states that he would rather work it th be near it. The rationality of his statement supported by the fact that the noise level approximately 8 decibels lower behind the han of the hammer than at the same distance either side.

The riveter is also exposed to intense no from 115 to 128 decibels. On the basis th the noise is greatest on the other side of the pl from the riveter, the "rivet bucker," who hand the small pneumatic hammer, would be expec to receive the greatest amount of noise. Ho ever, the metal these men are working on is s compared to the cold plate. The amount of no produced by these operation varies with temperature of the rivet. As the rivet cools loudness increases. Thus the noise produced the rivet hammer is substantially less than th produced by the chipping hammer while the ri is red hot and approaches it as the rivet rounded off.

The use of compressed air offers a hazard hearing. At least 1 case of total disability hearing as a result of the noise produced throu this medium has been reported. The noise var with the poundage of pressure, the size of opening, the distance from the opening and nature of the release, as from a sudden break the hose.

THE COURSE OF LOSS OF HEARING FRO NOISE IN INDUSTRY

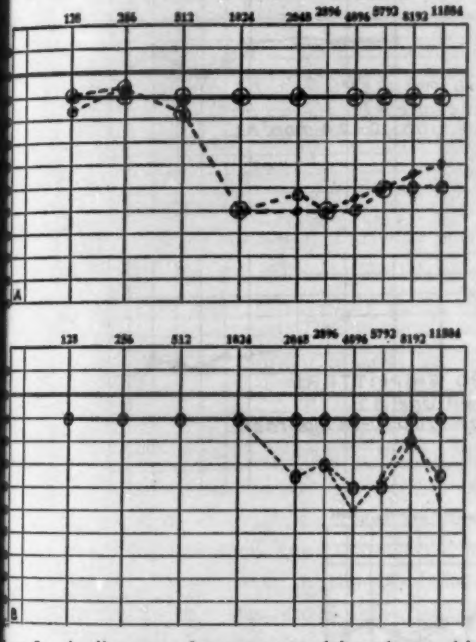
One hundred audiometric tests were[7] ma at the preemployment clinic of a large shipya to obtain men with perfect hearing who we going into noisy occupations. This testing w done with the idea of retesting their hearing af definite periods of exposure to noise to determ any changes incurred. The men selected we mainly Negroes. They had no past history exposure to noise, for the most part having be farmers, nor had they any history of disease the ear, nose or throat. Examination at t time revealed no evidence of such diseases.

6. Larsen, B.: Investigations of Professional Deafness in Shipyard and Machine Factory Labourers, Acta oto-laryng., 1939, supp. 36, p. 3.

7. Tests were made with the Maico audiometer.

After working seven hours at the chipping ool, exposed to 110 to 130 decibels of noise presentative group of men had a distinct loss earing, associated with a ringing in the ears a feeling of discomfort. The following day deficiency of hearing was discernible only by iometric examination and there was relief of associated symptoms. Chart 2A reveals this nge, which is related to nervous fatigue, adapon of the central nervous system and possibly sticity of the intra-aural muscles. Whether manent cochlear damage was inflicted by this ial insult provides interesting speculation.

After a period of one month there was a nite deficiency of hearing in the high fre-



hart 2.—Audiograms for men tested in a large shipd. The heavy lines indicate the level of hearing rded before the men were employed. In *A*, the en lines show the levels after they had worked n hours in the chipping school. In *B*, the broken show the levels recorded after the men had worked month in the chipping school.

ncy range. This was not materially affected a rest of one or two days. Examples of the phic recordings of hearing as tested with the iometer are shown in charts 2A and B.

Examination of chippers after a year or more exposure to noise revealed a similar, though e extensive, loss of hearing. The results, regated according to shipyard trades, are wn in chart 3. To what extent a person be exposed to noise and suffer only nervous gue, without actual destruction of hearing, is et undetermined. The main factors involved he process have been listed as follows:[4] (1) l time of exposure; (2) length of exposure period; (3) loudness of sound stimulus; (4) character of sound stimulus, continuous or interrupted; (5) frequency of the noise; (6) type of space (environment); (7) protective devices used; (8) age (older persons probably being more susceptible and having less recuperative power); (9) previous aural disease, and (10) constitutional factors, such as differences in sensitivity. The question is what duration of exposure is hazardous at a given intensity of noise. C. C. Bunch stated that a level of 80 to 90 decibels was hazardous.[8] No doubt this is true with extensively prolonged exposure. From a practical viewpoint, consideration of the level from 100 decibels up provides a limited group not too large for preventive action by management; protection instituted for workers exposed to these levels of noise would be a great step forward. Office employees, working at a level of noise of 55 to 70 decibels, are more frequently provided with measures for the control of noise than men working at a level of 110 to 120 decibels.

In the interest of the constant demand for increased production and speed of work, it appears that workers in a constant level of sound of 100 to 110 decibels or over should be given the benefit of the concerted action of management and safety engineers in a program for the control of noise. Certainly, workers with pneumatic hammers in confined areas would be able to work for longer periods and with more effectiveness if the distraction of intense noise were removed or diminished.

REMEDIES

The treatment of the problem of noise in industry lends itself to several methods of approach.

The working environment frequently is most easily modified.[9] This is true in such work as the testing of motors. The motor can be placed in an acoustically treated room and observation carried on outside from a control booth. Dry grinding and certain hammering procedures lend themselves to isolation either individually or collectively in an acoustically treated booth or room.

Improved methods of manufacturing and the redesigning of machinery provide the best means for control of noise. An example of improvement in procedure as a means of eliminating noise is the use of spot welding or of the multiple riveting machine for small rivets in place of several singly operated pneumatic hammers in work on fuselages. Intensely noisy hammering procedures frequently give way to methods involving

8. Bunch, C. C.: Conservation of Hearing in Industry, J. A. M. A. **118**:588 (Feb. 21) 1943.

9. McCord, C. P., and Goodell, J. W.: The Abatement of Noise, J. A. M. A. **123**:476 (Oct. 23) 1943.

slow hydraulic pressure and changes in the hardness of the steel.

Maintenance of machinery is a factor in the elimination of noise. A worn bearing or a flat wheel can be remedied.

In the shipbuilding industry and in certain branches of the aircraft industry the environment and the methods are not amenable to acoustic treatment. In these instances it is important that men forced to work at a high level of noise be supplied with efficient ear stoppers. It is well known that the worker's judgment as to whether he needs acoustic protection is unreliable. In many instances the unreliability of the man's judgment is due to the already existent loss of hearing, which provides some degree of cushioning.

Cotton is an old standby and reduces noise from 10 to 15 decibels, which in most cases still leaves the worker exposed to a hazardous level of noise. Rubber ear stoppers lower noise from 20 to 30 decibels. The difficulty lies in getting the workers to wear them over long periods. A plug molded so that it will navigate the initial sharp angle from behind the tragus into the opening of the canal and still efficiently occlude sound causes points of pressure to develop, with symptomatic reactions.

The problem has been to have ear stopp which not only eliminate the hazards of inte noise and prevent the entrance of fore material, but also are so acceptable to the wor that he will voluntarily make use of them long and repeated periods. The development such a stopper is most important. Modi plastic lucite ear molds are being used succe fully.[10]

COMMENT

In view of the beginning influx of cases occupational deafness requiring settlement, it essential from the viewpoint of all concerned t the possibility of a noise hazard in any plant given thorough attention. I believe that the so tion of this problem involves not only a surv of the level of sound but audiometric exami tion of exposed workers. In each such instan this procedure is useless and unfair to manag ment if executives are not made fully aware only from the aspect of their prime inter today, increased production) of the necess of taking active measures based on speci recommendations.

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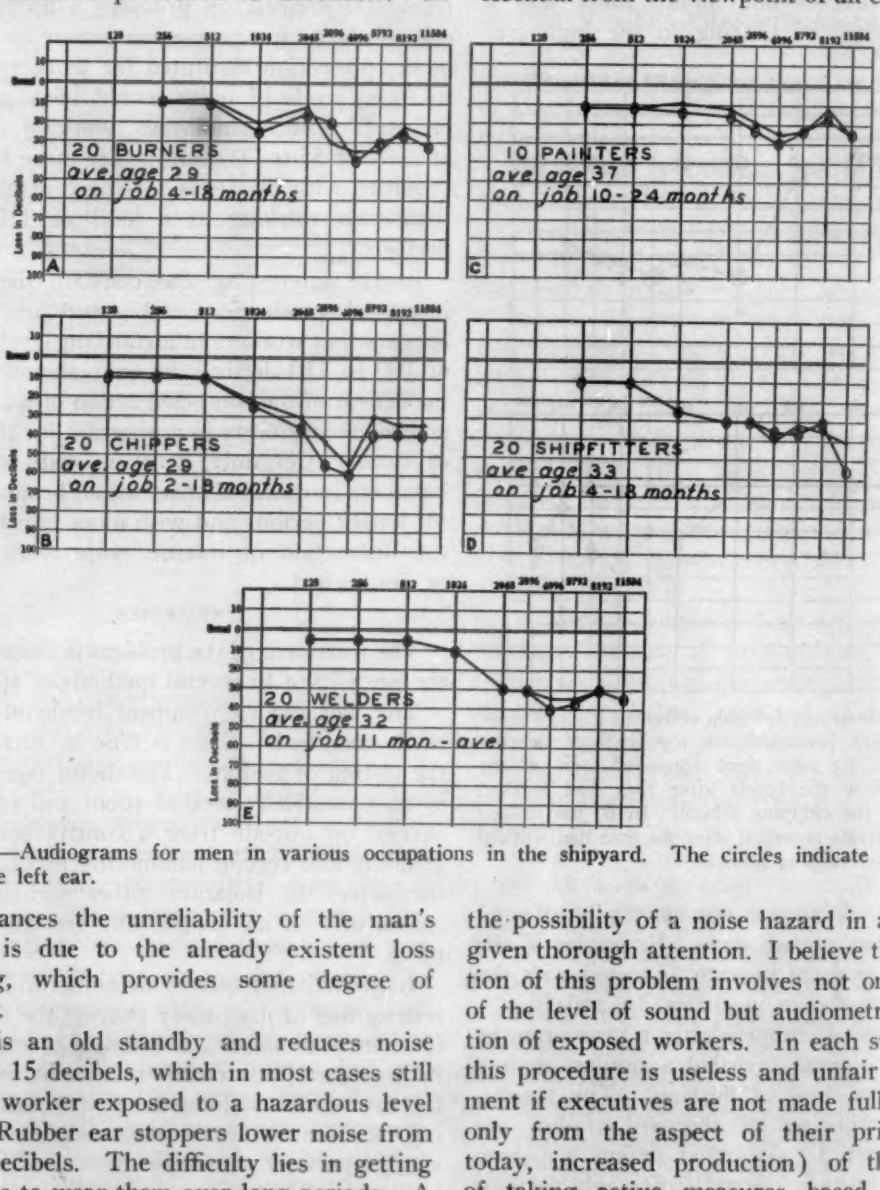

Chart 3.—Audiograms for men in various occupations in the shipyard. The circles indicate the right e the dots, the left ear.

10. McCoy, D. A.: Prevention of Ear Disability Industry: Report on the Use of a Plastic Mold, J. M. A. **121**:1330 (April 24) 1943.

EVALUATION OF SUBMUCOSAL SHRINKING METHODS FOR CHRONIC NASAL OBSTRUCTIONS

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In outlining shrinking methods for the treatment of perennial nasal obstructions we should like to emphasize that in cases of inflammatory or allergic disease of the nasal mucosa a causative factor or an immunobiologic basis should not be considered. Therefore, only those patients are selected for a shrinking treatment of the nasal mucosa on whom tests have first been made for nasal allergy and the reaction found to be negative or in whom clinical findings have excluded a sinus disease. The patients tested for allergy are those with "boggy" mucosa, judged into the group of patients with vasomotor rhinitis or with muscular dystonia of the cavernous bodies; in the group examined for sinus disease are found true hyperplasia and hypertrophy of the mucous membrane, both causing recurrent or permanent attacks of nasal tumescence.

Various methods for reducing the size of the turbinates have been recommended. Since surface applications of chemical astringents and caustics, as well as surface electrocoagulation, have been rejected for well known reasons, submucosal reductions as used in the obliterating treatment of varicose veins and submucosal diathermy have been introduced.

In addition to the reduction in size, the most desirable effect is attained in preservation of the epithelial linings and absolute avoidance of adhesions between the turbinates and the septal mucosa.

In the course of injection treatments we have learned that one cannot be indifferent to the amount of solution injected under the mucosa and into the cavernous bodies.

The cavernous bodies of the nose, in contrast to the true corpora cavernosa of the sex organs, are enlarged intercalated venous spaces without any direct communications with the arteries. The structure of the enlarged vascular spaces enables them to transmit a large amount of fluid into the body without any of the fluid being arrested at the place of injection. In fact, the inferior turbinate often has served as a conveyance for liquid drugs, especially when the veins in other structures could not be used as in burns or cutaneous disease.

To avoid escape of the injected fluids into other parts of the body, we have employed a simple technic which will be described later.

During the last two years more than 100 patients have been treated with injections or submucosal diathermy at the Illinois Eye and Ear Infirmary in Chicago.

The indications for the treatment, as mentioned, have been definite in regard to the kind of nasal obstruction in chronic engorgement of the mucosa of the inferior turbinate, with negative findings for allergy or sinusitis and with routine responses to shrinking solutions of epinephrine or ephedrine. In these cases we found the injection treatment most favorable. As the agent to be injected we have used sylnasol, which is a 5 per cent solution of the sodium salts of certain of the fatty acids of the oil extracted from a seed of the psyllium group, to which 2 per cent benzyl alcohol is added for its anesthetic effect. The preparation has recently been recommended by Thacker.[1]

In cases of chronic blockage of the nose from thickening of the mucosa with a negative response to the epinephrine group, submucosal electrocoagulation gave more satisfactory results. In cases with true hyperplasia and hypertrophy the submucosal destruction of the tissue should be more extensive, and this could be better accomplished and controlled by electric current.

HISTOLOGIC OBSERVATIONS

For further observation of the action of the introduced agents and of electrocoagulation in the inferior turbinate we have studied series of biopsy specimens taken repeatedly at different times after injection and after electrocoagulation.

After Injection.—In a specimen removed several hours after injection of the sclerosing agent we could observe coagulation necrosis of the

1. Thacker, E. A.: The Treatment of Chronic Obstructive Rhinitis with Submucosal Injections of Sodium Psylliate, Ann. Otol., Rhin. & Laryng. 49:939, 1940.

tissue strongly marked around the punctured canal in an area about 1 to 2 mm. wide, affecting partly the lymphoid stratum and destroying the deep glandular and vascular layers. The mucosa with the basal membrane and the superficial connective tissue layer showed here and there a concomitant edema with enlargement of the lymphatic canals.

At the walls of the corpus cavernosum the endothelial cells were deformed and partly separated from the surrounding muscular fibers. The lumens of the affected vascular spaces were filled with a clot of red blood cells.

In a specimen removed forty-eight hours after the injection we could see deposits of fibrin in the vessels and in the interstitial capillaries. The injected area showed signs of an exudative phase caused by the aseptic inflammation, marked by an increase in the white blood cells and the fibroblasts.

An eight day specimen showed obliteration of the vascular lumens; obturating thrombi were penetrated by connective tissue cells preparing organization and shrinking of the tissue. The media of the vessels and the muscle fibers of the corpus cavernosum were homogenized with staining signs of hyaline degeneration. The destroyed glands were replaced by round cells and fibroblasts (fig. 1).

In a section of the mucosa eight weeks after the injection we found general sclerosis of the connective tissue layers. The affected glandular layer was replaced by connective tissue, and the vascular stratum was obliterated completely. The mucosa and the basilar membrane were in normal condition.

After Coagulation.—The method used in electrocoagulation of hypertrophic turbinates was first introduced by Beck.[2] The histologic changes were analogous to those after injection with more extensive tissue changes.

In a section removed soon after the needle was inserted we found the epithelial cells, the membrana limitans and the superficial connective tissue layer intact.

The tissue around the punctured and electrocoagulated area showed a pale blue discoloration with hematoxylin-eosin stain.

The connective tissue fibers appearing normally as red bundles became a swollen pasted mass; the scarcely visible nuclei appeared pyknotic and fragile. The walls of the vessels were thickened; their deformed lumens were partly empty and partly filled with fresh thrombi. The adventitia and the media became a homogenized swollen ring.

At the periphery of the coagulated area the tissue changes were less intensive. In the hyperemic enlarged vessels one could see leukocytes accumulated in the marginal positions and few hemorrhages in the edematous surrounding. Striking changes were observed in the glandular layer. Mucous and serous glands were highly deformed, and their lumens were filled with

Fig. 1.—Obturating thrombosis and organization of the vascular layer.

2. Beck, J. C.: Pathology and Intramural Electrocoagulation of the Inferior Turbinate, Ann. Otol., Rhin. & Laryng. **39**:349-363, 1930.

tted mucous and cellular debris. The cyto-sm of the glandular cells appeared puffed up d interspersed with minute vacuoles. The nted nuclei changed to flat, elongated struc-es and were pasted up with the coagulated ss in the lumens (fig. 2 *A*).

The formerly delicate glandular membrana ppria became thick and fused with the sur-nding connective tissue. The vascular layer

Fig. 2.—*A*, deformation of the mucosal glands; *B*, rked thickening of the glandular membrana propria.

owed degeneration of the endothelial lining d thrombi throughout the affected area (fig. B).

Sections presenting the effects of later periods electrocoagulation showed rich cellular infil-tion and subsequent organization to connec-e tissue replacing both the vascular and the ndular layer but leaving the mucosal epi-elium unchanged.

TECHNIC OF INJECTION

Injections should first be made into the anterior third of the inferior turbinate. After cocainizing the tip with a 5 per cent solution of cocaine, the back part of the nasal cavity below the middle meatus is packed with cotton saturated in a 0.5 per cent solution of ephedrine. These packs produce a partial mechanical anemia preventing escape of sclerosing agents.

The injection should be made with a very sharp thin pointed needle and directed toward the corpus cavernosum. After the needle has been inserted 1 cm. deep, the mucosa should be lifted from the periosteum and about 1 cc. of the drug slowly injected. As soon as the needle is removed a cotton pledget is pressed over the punctured area, and after the bleeding has stopped the cotton packs are removed. A fortnight later a second injection can be made, starting from the middle of the inferior turbinate.

EVALUATION

Observations made after treatment convinced us that the submucous methods of shrinkage do not produce any inflammatory reaction or fibrinous secretions in the nasal cavity as seen after surface coagulations with chemicals or galvanocautery. In about 50 per cent of the patients one submucous injection or diathermic coagulation was sufficient to increase the air passage and give permanent relief.

Undoubtedly all coagulants, the submucous ones as well as those applied to the surface, may cause, in addition to the local alteration of the tissue, transitory reactions in the body, which often resemble an allergic shock. These reactions occur especially in patients who were previously treated with injections. However, these fugitive reactions are probably due to reincorporation of the injecting agents which act as heterogeneous protein in the sense of an anaphylactic constituent. The reactions subside without treatment, but on two occasions a sudden fall of leukocytes was encountered.

On the other hand, it is believed that the striking successful results lie in the rapid destruction of the cells and in the increased reabsorption of the decayed cellular proteins which act as a desensitizing factor.

SUMMARY

Submucous injection of sylnasol and submucous electrocoagulation were used to reduce nasal obstruction in a series of 100 patients. Postoperative bleeding did not occur. Therefore, no packing of the nose or other special after-care was necessary. The epithelium and the membrana propria remained untouched. The inner production of fibrous tissue was proportioned and satisfactory. The general reactions were transitory and did not receive special attention.

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MUSCLES AND CARTILAGES OF THE NOSE FROM THE STANDPOINT OF A TYPICAL RHINOPLASTY

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Rhinoplasty in its early stage had a purely reparative indication for traumatic disfigurement. Since Joseph developed the basic principles of intranasal technic, plastic surgery of the nose has been practiced frequently for preformed cosmetic reasons alone. The experience of the present day teaches that a good cosmetic result can be achieved only by restoring perfect physiologic function of the nose. Therefore the physiology and the morphology of the nose share equally in importance.

The trend of modern plastic surgery is to avoid damage to structures not directly involved in the operative procedure. This principle applies especially to the fine muscles of expression which are situated on or about the nose. It has been sinned against frequently in the past. Any separation in any layer not absolutely necessary for the surgical attack will result in a layer of scar tissue with impairment of function of the organs adjacent to the layer in which the scar develops. This is especially true if the subcutaneous separation of the entire skin of the nose is practiced. This is unnecessary. The only elevation which is really necessary is the separation of the soft structures over the dorsum. It consists (1) of the separation of the soft structures of the upper cartilaginous vault above the perichondrium of the upper lateral cartilages and (2) the separation of the soft structures of the osseous vault below the periosteum. The result of a poorly executed rhinoplasty is that the delicate structures, i. e., the fine muscles and tendons involved in facial expression about the nose, are immobilized between layers of scar. In spite of good morphologic results, the nose stands on the face like a piece of marble, without taking part in the fine modulations of facial expression. In addition, nasal breathing may be impaired. In this study the anatomy of the muscles and cartilages of the nose was reconsidered from the standpoint of plastic surgery.

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Read at the meeting of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, in New York, Feb. 25, 1944.

The question was investigated whether the usual endonasal incisions do not interfere with the function and the direction of contraction of the muscles and with nasal respiration.

ANATOMY OF THE MUSCULATURE IN RELATION TO FUNCTION

The soft parts of the nose, especially the lower cartilaginous vault, can be drawn upward (cranially) or downward (caudally) by muscular action. The nostrils can be compressed passively by the preponderance of the pressure of the outer air over the inspiratory negative pressure in the vestibule and can be actively dilated by the dilator muscles during deep inspiration.

It seems probable that the impulses which emanate from the breathing center also affect the muscles of the alae nasi (Mink[1]). With increasing widening of the thorax, a contraction of the musculature of the alae nasi sets in.

The musculature of the face participates in different functions: the intake of food, chewing, speech, functions of the eyes and the nose and facial expression.

The highly differentiated muscles of the face arise from the periosteum of the bony skeleton and insert into the elastic skin, mucous membranes, fasciae or the perichondrium of the cartilages. The individual mimetic muscles lack well defined fascia, in contrast to the skeletal muscles. For this reason, it is possible that a very small portion of a muscle may contract independently of the rest of the muscle.

The muscles which play a main part in the movement of the lower cartilaginous vault and the skin of the nose can be classified into four groups:

1. Elevator muscles, which shorten the nose and dilate the nostrils.

(*a*) The musculus procerus (fig. 1*7*).

(*b*) the caput angulare musculi quadrati labii superioris (fig. 1 *1,2,3,4*).

2. Depressor muscles, which lengthen the nose and dilate the nostrils.

1 Mink, P. J.: Physiologie der oberen Luftwe Leipzig, F. C. W. Vogel, 1920.

(*c*) the pars alaris musculi nasalis.

(*d*) the musculus depressor septi nasi (fig. 11).

3. Compressor muscles, which lengthen the nose and narrow the nostrils.

(*e*) the pars transversa musculi nasalis (fig. 8).

4. Specific dilator muscles of the nostrils (fig. 10).

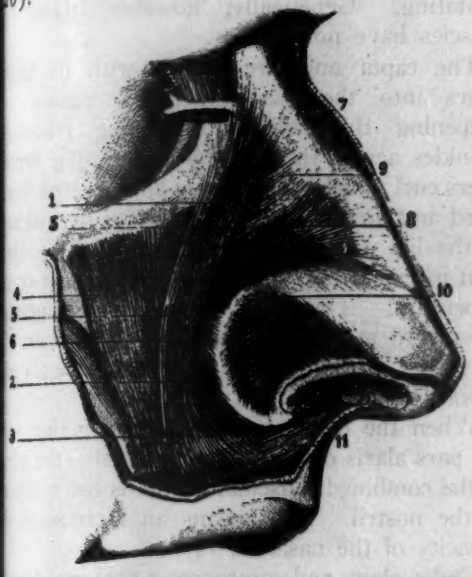

Fig. 1.—External muscles of the nose from P. C. Appey (Traité d'anatomie descriptive, Paris, V.-A. Delahaye & Cie, 1889): *1*, caput angulare musculi quadrati labii superioris; *2*, nasal fibers of *1*; *3*, labial fibers of *1*; *4*, *5* and *6*, caput infraorbitale musculi quadrati labii superioris; *7*, musculus procerus; *8*, pars transversa musculi nasalis with aponeurosis; *9*, musculus nasalis; *10*, musculus dilatator; *11*, musculus depressor septi nasi.

The majority of the nasal muscles, independent of their lengthening or shortening effect, assist in widening the nasal opening. This proves the significance of unobstructed nasal breathing for the preservation of life. There is only one nasal muscle on each side which actively narrows or compresses the nostril: the pars transversa musculi nasalis. The pars transversa musculi nasalis lies over the upper cartilaginous vault of the nose, above the wings. It forms with its mate from the other side a kind of sphincter around the alae, overlying the bridge of the nose. The middle part of this sphincter consists of a fibrous aponeurosis which is adherent to the skin but is not closely attached to the underlying cartilaginous structures. For this reason, the separation of the soft tissues over the upper cartilaginous vault is best done in this situation.

There is a significant difference in the structure of the connective tissue of the lower and the upper cartilaginous vault. In the former the connective tissue is so tight that skin, muscle and cartilage are nearly inseparable, forming one movable unit. In the upper cartilaginous vault the skin together with the aponeurosis can slide upward and downward on the cartilage.

The pars transversa musculi nasalis arises above and lateral to the incisive fossa of the maxilla. Some of its fibers insert into the skin of the ala and into the skin of the nasolabial fold. The major part, however, joins the aponeurosis which connects both sides. Muscle fibers and aponeurosis cover the upper lateral cartilages and the adjacent membranes of connective tissue. A bursa facilitates the displacement between the aponeurosis and the upper lateral cartilages. The pars transversa forms with its fellow a sling.

It is obvious that when this muscle contracts the aponeurosis together with the adherent skin slides downward toward the lower cartilaginous vault. During inactivity of the nasal muscles the lower lateral cartilages overlap the upper lateral ones at the inferior (caudal) border of the latter. Between the two rows of cartilages there is a small intercartilaginous ligament in which two or three fine pieces of cartilage are enclosed [2] (fig. 2*A*): It is my belief that these minute pieces of cartilage may act like a ball-bearing mechanism making possible the rolling of the overlapping cartilages. The intercartilaginous ligament will be stretched by the contraction of the pars transversa musculi nasalis. In this way the lower cartilaginous vault is abducted from the upper cartilaginous vault. This stretching of the soft structures lengthens the nose.

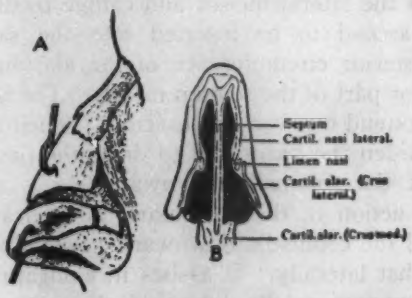

Fig. 2.—*A*, upper and lower lateral cartilages with the intercartilaginous ligament in which three fine pieces of cartilage are enclosed.

B, Frontal section of the nose, close to the piriform aperture. The septum and upper lateral cartilages are seen to be one unit. The lateral crus of the alar cartilage overlaps the caudal border of the wings of the septum. At the limen nasi a fine piece of cartilage is visible, forming a kind of ball-bearing mechanism.

2. Meriel, P.: Recherches anatomiques sur certains cartilages du nez, Arch. d'anat., d'histol. et d'embryol. 4:149, 1925.

The upper lateral cartilages are wings of the septum just like the wings of the sphenoid bone. They are fixed parts of the septum (fig. 2 *B*). The real movable part of the nose is the lower cartilaginous vault. The nose can be lengthened or shortened only as much as the intercartilaginous ligament allows (fig. 2 *A*).

The pars transversa musculi nasalis is especially active in the production of certain sounds. It has its greatest activity in the production of the consonant sounds "p" and "b." Here the lips are burst apart by the air pressure behind them. The muscle is less vigorous in the production of the humming sound "m," and it shows only feeble action in the sounds "f" and "v." Active contraction of the pars transversa is visible in the sound "oo," in whistling and in the crying of newborn infants. During the contraction of the muscle, the nose appears pinched or flattened (Lightoller [3]).

The pars transversa musculi nasalis elongates the nose by rolling the lower lateral cartilages downward and compresses the vestibule. The effect of this activity is to block the airway more or less effectively. The diminution of the external orifice assists in maintaining the air pressure within the cavum oris. Some persons are able to constrict their nares with great force, especially in order to avoid the inhalation of dust or a bad odor, which might irritate the mucous membranes of the nose mechanically or chemically.

The pars alaris musculi nasalis, a small quadrangular muscle, originates from the alveolar juga of the lateral incisor and canine teeth. Its fibers ascend to be inserted into the skin of the posterior circumference of the ala, and the posterior part of the septum mobile. The muscle fibers extend in a vertical direction. Their action is to widen the nares and to draw the posterior part of the columella downward.

The action of the pars alaris is to draw the ala and the columella downward (caudally) and somewhat laterally. It assists in elongating the nose, supporting the action of the pars transversa. However, in widening the nares it acts antagonistic to the action of the pars transversa.

The pars transversa and the pars alaris musculi nasalis may act in conjunction with another important nasal muscle, the nasal portion of the caput angulare musculi quadrati labii superioris. The caput angulare originates phylogenetically from the medial part of the musculus orbicularis oculi. Its head, therefore, either may be fused with the orbital sphincter at the medial canth of the eye or may arise as a separate divisi from the processus frontalis of the maxilla. origin can extend far outward at the inferior r of the orbit, below the musculus orbicula oculi. The caput angulare sends down a na fasciculus which covers the insertion of the pa transversa musculi nasalis. Both muscles m form a common muscle plate, the fibers int digitating. Genetically, however, these t muscles have no relation.

The caput angulare inserts with its later fibers into the nasolabial fold, raising a deepening this fold and causing transver wrinkles across the osseous vault. The centr fibers curl around the ala nasi and proceed dow ward and forward to the margin of the red ar of the lip. The most posterior of these fibe gain insertion into the mucous membrane of t upper lip. The most medial fibers proce downward and forward to be attached into t ala nasi. The caput angulare draws the ala na upward cranially.

When the caput angulare acts together wi the pars alaris of the musculus nasalis, the effe of the combined muscular action is the widenin of the nostril. This means an increase of t capacity of the nasal airway.

Embryology and comparative anatomy demo strate that the musculus procerus develops fr the caput angulare musculi quadrati labii supe oris. There are tendinous connections with t musculus frontalis, but the musculus procerus not simply the continuation of the frontal musc toward the nose. Genetically the two musc have nothing in common, and in action each just the reverse of the other: The musculu frontalis draws the skin of the forehead upwar the musculus procerus draws it downwa (Braus [4]). The musculus procerus (the nam means the slim or slender) has its fixed poi of origin from the periosteum of the nasal bo and from the aponeurosis of the pars transver musculi nasalis. Frequently both origins appe together. It runs upward in a vertical directi (the most lateral fibers nearly horizonta toward the forehead and is in some cases so wi that the skin of the root of the nose appea broader than the bone. It has its insertion the skin above the glabella between the ey brows and in the skin of the inferior part the forehead, spreading sometimes fanshape Because of this fanlike extension it is sometim called musculus pyramidalis. Often its size the right side differs from that on the left si and frequently it is fused with the mate of t

3. Lightoller, G. H. S.: Facial Muscles: The Modiolus and Muscles Surrounding the Rima Oris with Some Remarks About the Panniculus Adiposus, J. Anat. **60**:1, 1925-1926.

4. Braus, H.: Anatomie des Menschen, Berl Julius Springer, 1921, vol. 1.

er side to form a common plate. It has an erage length of 12 to 15 mm.

The musculus procerus forms a bridge over e frontonasal angle. During its active conction the muscle causes one or more transrse wrinkles of the skin across the root of the se and so gives an expression of menace.

sum of the nose. In many cases the musculus procerus travels down to the middle of the dorsum, ending in the skin, in the perichondrium of the upper lateral cartilages and in the periosteum of the nasal bones (fig. 3 *A*). Sometimes the musculus procerus as an imposing unbroken muscle covers the entire lateral side

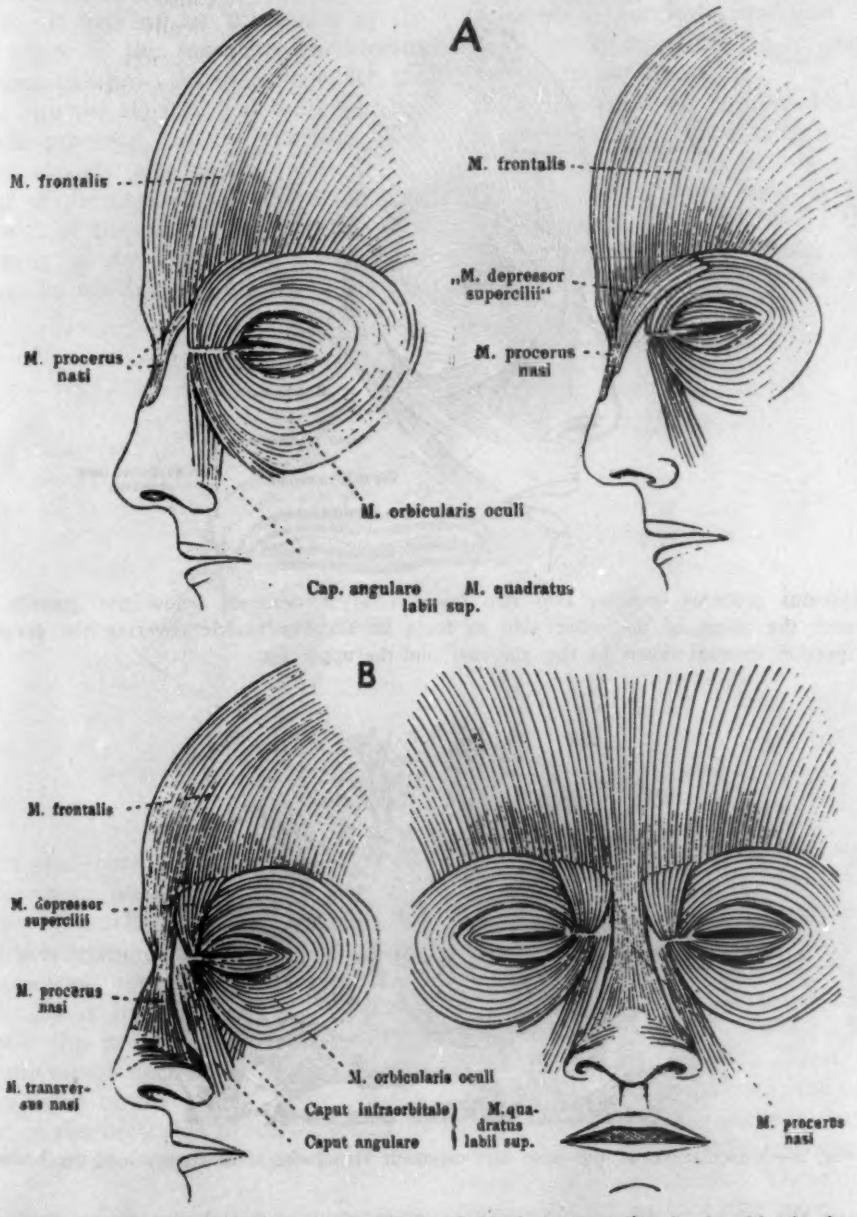

Fig. 3.—*A*, musculus procerus extending to the middle of the dorsum, ending in the skin, in the perichondrium the upper lateral cartilages and in the periosteum of the nasal bones. *B*, unbroken musculus procerus covng the entire side of the nose and extending down as far as the ala nasi.

Huber [5] described cases in which the parts on th sides fuse with one another and form an ygous muscle which runs along the bony dor-

of the nose and extends as far downward as the ala nasi (fig. 3 *B*). Finally there are other cases in which the musculus procerus arises broadly fused with the frontal muscle. As it descends below the glabella, it divides into two

5. Huber, E.: Ueber die Morphologie des M. prorus nasi des Menschen, Anat. Anz. **51**:302, 1918.

fasciculi. The medial bundles fuse with the fibers of the other side to form an azygous muscle covering the dorsum nasi. The strong lateral portion, however, extends downward to the ala nasi and the upper lip (fig. 4).

The morphologic differences in the musculus procerus may serve to explain the action of the muscle. In its simplest development it draws

One gets an instructive picture (Eisler[6]) observing the musculature of the nose fr behind after removal of the bones (fig. 5). O sees the common plate of the musculus proce and fibers decussating from one side to t other. In the same figure the direction of t fibers of the musculus nasalis, which circles t nares, should be observed carefully. They c

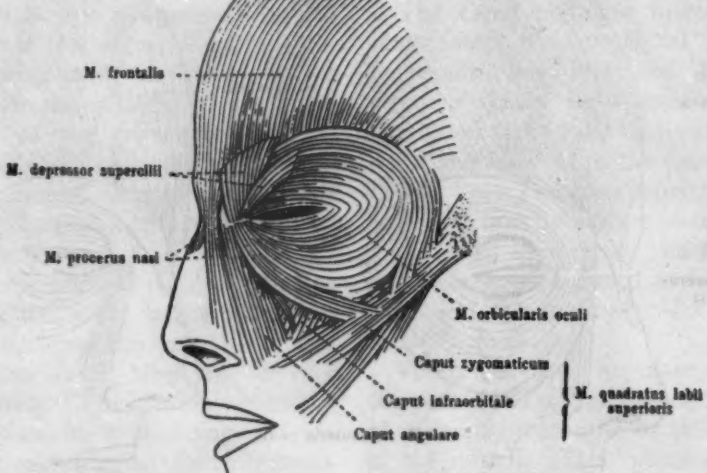

Fig. 4.—Musculus procerus dividing into two fasciculi as it descends below the glabella. The medi bundles fuse with the fibers of the other side to form an azygous muscle covering the dorsum nasi. T strong lateral portion extends down to the ala nasi and the upper lip.

Fig. 5—Musculature of the nose and adjacent structures after removal of the bones.

the skin above the glabella downward, causing transverse wrinkles at the root of the nose. The fibers extending to the ala raise the ala and draw the skin of the cartilaginous nose upward, shortening the length of the nose.

These two different directions of muscle action find their highest development morphologically in independently functioning separate groups of muscle fibers.

circularly around the external nasal opening These observations gain importance from th standpoint of endonasal incisions.

The musculus procerus is called by Virchow "depressor glabellae." This undoubtedly is on

6. Eisler: Die Muskeln des Stammes, von Bardel ben, K.: Handbuch der Anatomie des Menschen, Je Gustav Fischer, 1912.

7. Virchow, H.: Die Muskulatur der Nase, An Anz. 58:137, 1924.

nction of the muscle. Its second function is etermined by its close connection with the oneurosis of the pars transversa musculi asalis. When the latter muscle contracts the oneurosis slides a little downward and retches the musculus procerus passively.

I have arrived at the conclusion that the active ontraction of the musculus procerus may raise he skin of the cartilaginous vault and the tip f the nose. It acts as an antagonist to the epressing effect of the musculus transversus asi (musculus nasalis). In cases in which the bers insert into the ala nasi and the upper lip, he musculus procerus has the function of a ilator or an elevator alae nasi.

The tonus of the musculus procerus together ith the tonus of the other nasal muscles and he overlapping of the lower lateral cartilages re the means by which the nares are kept open. It is known that persons recently dead show conspicuously long noses because of the lack of this tonus (Hovorka [8]).

From these anatomic considerations it will be understandable that severance of the musculus procerus eliminates its elevating effect on the nasal tip and the preponderant action of the musculus transversus nasi tends to depress the cartilaginous vault of the nose. Therefore the preservation of the musculus procerus seems to be important for the upward fixation of the nasal tip.

In long noses or noses with a big hump, it seems obvious that the musculus procerus may be elongated according to the deformity. After the shortening of the nose there still remains an elongated musculus procerus. Especially in cases in which this muscle is well developed, the shortening of the cartilaginous nose approximates the points of origin and insertion of the musculus procerus. The fibers will then appear compressed, filling out the frontonasal angle. In such cases the forehead and the nose form a straight line, which is an unpleasant cosmetic result. In order to avoid such undesired filling out of the nasal frontal angle, one can try to resect longitudinal strips of muscle tissue.

The muscles of the ala nasi should not be neglected in surgical correction of the lobule. Their activity regulates the resistance of the air flow in the air passages.

Laterally from the nasolabial sulcus there are the insertions of the caput angulare and the caput infraorbitale of the musculus quadratus labii superioris. It is interesting to know that the caput angulare is derived from the orbicularis oculi; the caput infraorbitale, however, is a descendant of the sphincter of the mouth.

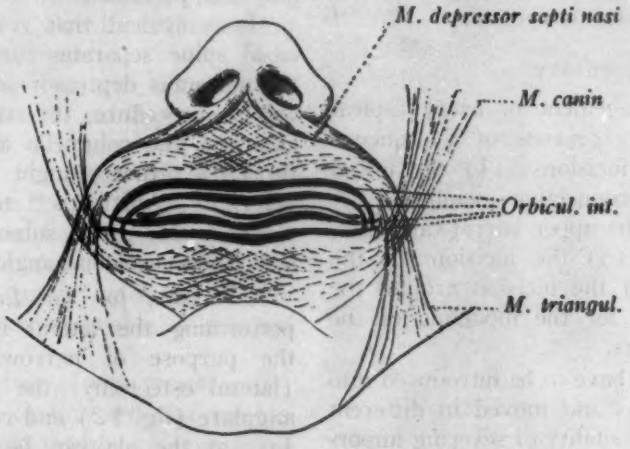

Fig. 6.—Musculus depressor septi nasi.

Medially from the nasolabial sulcus one observes the musculus dilator of the nares. This is a thin triangular muscle which lies on the side of the wing of the nose. One can feel the contraction of this muscle if one takes the alae between two fingers. Its muscle fibers originate sometimes from the upper margin of the piriform aperture.

Some authors mention often a small thin muscle at the tip of the nose: the musculus apicis nasi, which dilates the anterior part of the nares. This muscle is of little importance from the standpoint of rhinoplasty. These dilators are real "primary" dilators of the nares, enlarging the circumference of the nasal apertures. The others mentioned are "secondary" dilators, because they widen the alae by drawing from above or from the side. The nasal aperture can be further enlarged from below by contraction of the musculus depressor septi nasi (fig. 6). This muscle represents the most cranial fibers of the musculus orbicularis oris. The fibers insert into

8. von Hovorka, O.: Die äussere Nase, Vienna, A. Hölder, 1893.

the columella (septum mobile nasi) and into the perichondrium of the cartilaginous septum. They can reach the medial crus of the lower lateral cartilage and thereby the tip of the nose. The bundles are attached to the entire medial half of the dorsal border of the nasal aperture (insertio nasalis). Besides this insertio nasalis there is an attachment to the bone, especially to the anterior nasal spine (insertio ossea).

In contrast to the action of the pars transversa musculi nasalis, which pushes the entire cartilaginous vault of the nose downward, the musculus depressor septi nasi draws down the columella, the tip of the nose and the dorsal border of the nostril.

The muscles of the nose vary considerably in the extent of their development. Occasionally authors have found a longitudinal muscle strip, the musculus anomalus nasi. This lies at the lateral side of the bony nose in a region where mostly no muscular tissue is present.

RHINOPLASTY

The operative management of every typical rhinoplasty (Fomon [9]) consists of a sequence of several endonasal incisions: (1) the initial incision through the connective membrane between the lower and the upper lateral cartilages, (2) the transfixion, (3) the incision for the lateral osteotomy, (4) the incision around the margin of the lobule for the modeling of the lower lateral cartilages.

Special instruments have to be introduced into each of these incisions and moved in different planes. There is a possibility of severing important structures and creating undesired functional disturbances.

1. *Initial Endonasal Incision.*—Much harm can be done with the first endonasal incision through the fibrous ligament between the upper and the lower lateral cartilages. When the edge of the knife is held in a vertical instead of a horizontal direction, it may cut the muscle fibers of the pars transversa musculi nasalis or the aponeurosis which connects both sides. This may cause widening, a "decompression" of the cartilaginous vault. The separation of the skin from the upper cartilaginous vault should be done between the perichondrium of the upper lateral cartilages and the aponeurosis of the pars transversa. This can be facilitated by passively elevating the tip of the nose, pushing the aponeurosis on its bursa upward. After the elevation of the periosteum of the nasal bone on both sides, the soft tissues overlying the ventral and lateral nasal walls are freed. A button e knife is introduced through the left vestibul incision and the two planes of separation (abo the perichondrium of the upper cartilagino vault and below the periosteum of the na bones) are brought into one plane. During t sweeping obliquely downward across the dorsu until the tip of the knife appears through t intercartilaginous incision in the right vestibu care must be taken to direct the sharp edge the knife toward the bony and cartilaginou structures and not toward the overlying so tissues.

2. *Transfixion.*—The knife which transfix the nose cuts only through two thirds of t membranous septum. The remaining one thir extending to the anterior nasal spine is fr quently severed by straight Mayo scissors. some cases resection of the anterior nasal spi has to be performed.

It is evident that removal of the anteri nasal spine separates the osseous insertion the musculus depressor septi nasi. As a resu of this procedure, the nasolabial angle can l absent. The columella and the upper lip a proach a rather straight line. To correct th deformity, Aufricht's [10] technic can be recom mended. He uses a submerged suture to reco struct the nasolabial angle.

3. *Incision for the Lateral Osteotomy.*—I performing the lateral intranasal incision f the purpose of narrowing the nasal bon (lateral osteotomy) the insertion of the capu angulare (fig. 1 *2*) and caput infraorbitale (fig 1 *6*) at the ala can be injured. The tip the knife should be directed straight to the pir form aperture, incising the overlying mucos The line of the incision follows the margin of th piriform aperture. In extending this incisio downward one should remember the circula direction of the surrounding muscle fibers a demonstrated in figure 5.

4. *Incision for the Modeling of the Lowe Lateral Cartilages.*—A circumferential incisio is made through the vestibular skin along th lower margin of the lower lateral cartilage. Th overlying structures immediately above the per chondrium are separated with a knife or with sci sors. When the instrument stays on this plan the musculus dilator will not be injured.

While it appears comparatively easy to avoi damage to the muscles, another problem in rhino plasty is still to be solved. The lower edge o the upper lateral cartilages, i. e., the upper par of the ball-bearing mechanism, can be seen i

9. Fomon, S.: The Surgery of Injury and Plastic Repair, Baltimore, William Wood & Company, 1939.

10. Aufricht, G.: A Few Hints and Surgical Detail in Rhinoplasty, Laryngoscope **53**:317, 1943.

vestibule as a marked line, which is called limen nasi. It has been given the name of asal valve" by Mink.[11] The limen nasi together with the opposite section of the septum rms a narrow passage, the ostium internum Mink[11]). If one compresses one's own ostia erna a little, not completely, by pressing the lve areas between the finger and the thumb, ving, however, the actual alae nasi free, one tices how the latter perform an inspiratory pansion in an attempt to force the obstruction (Uddströmer[12]).

Joseph observed that often deformities move ly in an inward and outward direction, ore exaggerated during laughing. When a rson laughs, the dorsal attachments of the alae si are drawn upward and laterally. Through ver-like movement, the tip of the nose slides wnward. Thus, during laughing, an otherise straight nose can show a hump, usually of e cartilaginous vault. For the same reason e nose often appears longer.

The intercartilaginous ligament between the per and the lower lateral cartilages with its ll-bearing mechanism on one side and the rous tissue between the septal cartilage and e crus mediale of the lower lateral cartilage he septum mobile) on the other side act like ticular ligaments. Without this articulation e rolling mechanism cannot function. Therere muscular action becomes unable to lengthen e nose.

Unfortunately after two of the typical endonasal incisions (1 and 2) this articular mechaism is more or less destroyed by an immobilizg scar, which causes a kind of ankylosis.

11. Mink, P. J.: Das Spiel der Nasenfluegel, Arch. d. ges. Physiol. **120**:210, 1907.
12. Uddströmer, M.: Nasal Respiration, Acta otoryng., 1940, supp. 42, p. 3.

CONCLUSIONS

1. The upper lateral cartilages as part of the nasal septum and adherent to the nasal bones move only in an inward and outward direction.

2. The lower cartilaginous vault can be elevated or lowered by muscular action.

3. Two or three minute pieces of cartilage in the intercartilaginous ligament act like a ball-bearing mechanism making possible the rolling of the overlapping cartilages.

4. The width of the intercartilaginous ligament represents the maximum amount of lengthening or shortening of the nose.

5. There is only one pair of muscles which narrows the nostrils actively: the pars transversa musculi nasalis. It compresses the internal nasal opening, the limen nasi. All the other nasal muscles are partly or mainly dilators of the nostrils.

6. The action of the musculus procerus is antagonistic to that of the pars transversa musculi nasalis.

7. There is a potential danger of injuring physiologically important muscles and fibrous membranes through endonasal plastic surgery.

8. To avoid damage to the fine muscles of facial expression, the lines of incisions and the planes of separation of tissues should follow the directions of the muscle fibers and of the aponeurosis.

9. The separation of the overlying soft structures should be done as close as possible to the bone and to the cartilage.

10. How to preserve the ball-bearing and valvelike mechanism of the alae nasi should be investigated further.

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p_H OF THE NASAL MUCOSA MEASURED IN SITU

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In a consideration of the causation of the common cold the predisposing factors must be included in addition to the inciting agent or agents. However, these factors will not be well understood until more complete information pertaining to the physiology of the upper respiratory passages is available, although physiologic features such as ciliary action have already been thoroughly studied.[1] In addition, various properties of nasal secretions, including activity of the lysozyme [2] and viricidal action,[3] have been examined and have been shown to represent physiologic activities related to the defense of the nose against infections caused by bacteria and viruses. Other factors, such as air currents [4] and variations in the supply of blood,[4] have received inadequate attention.

Fabricant [5] has recently studied a physiologic property of the nasal mucosa, namely, its p_H, which might affect both nonspecific and specific defense mechanisms of the nose. He approached the problem directly by making the measurements in situ with the aid of a glass electrode. The observations reported by this author were somewhat unexpected, as they indicated that the p_H of the normal mucosa varied between 5.5 and 6.5, with marked changes during a short interval of time. Such fluctuations in p_H, especially with values as low as 5.5, might easily affect the bacterial and virus flora of the nose. Hence, as part of a study of physiologic factors which can influence resistance to infections of the upper respiratory tract, we have reinvestigated the p_H of nasal secretions measured in situ.

METHOD

In a determination of p_H by the use of the glass electrode, it must be so designed that its entire membrane is in constant contact with the material the p_H of which is being measured. Furthermore, there must be no source of potential between the indicator electrode and the reference electrode unless such source or sources of potential are constant and subject to correction. The method described by Fabricant did not seem to insure adequate contact of the glass membrane with the nasal mucosa. Also, the point of contact of the reference calomel electrode with the body was not sufficiently close to the point of contact of the glass electrode to avoid extraneous sources of potential which seriously affected the measurements of p_H.

A somewhat different system of measuring the p_H of nasal mucosa in situ was therefore devised in an attempt to avoid these two possible sources of errors. A special glass electrode was designed [6] that a flat glass membrane closed the end of the electrode tube, which was beveled at an angle of approximately 60 degrees. With this type of electrode it was relatively easy to make satisfactory contact with the mucous membrane of either the medial or the lateral wall of the nasal cavity. The electrode was applied and was held by hand for the single determinations or by a supporting clamp attached to a band around the patient's head when measurements were made over a longer interval. A wick saturated with isotonic solution of sodium chloride was placed on the floor of the nose and connected to the calomel reference electrode by a bridge of sodium chloride solution. The reference electrode was in contact with the lip of the patient in the manner described by Fabricant. A Beckman p_H meter was used. If the electrode was held in good contact with the mucosa constant readings were always obtained. In some instances repeated readings were taken on the same person

This study was aided by a grant from the Kresge Foundation.

From the Hygienic Laboratory, University of Michigan.

1. Proetz, A. W.: J. Laryng. & Otol. **49**:557, 1934.
2. Fleming, A.: Proc. Roy. Soc. Med. (Sect. Path.) **24**:1, 1932. Hilding, A.: Changes in Lysozyme Content of Nasal Mucus During Colds, Arch. Otolaryng. **20**:38 (July) 1934.
3. Burnet, F. M.; Lush, D., and Jackson, A. V.: Brit. J. Exper. Path. **20**:377, 1939. Francis, T., Jr., and Brightman, I. J.: Proc. Soc. Exper. Biol. & Med. **48**:116, 1941.
4. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941.
5. Fabricant, N. D.: Significance of p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:150 (July) 1941.
6. Made for us by National Technical Laboratories, South Pasadena, Calif.

intervals of from one to five minutes for riods of from fifteen minutes to an hour.

It was found that a poor or an incomplete ntact with the mucosa by either the glass ctrode or the wick of the reference electrode nsistently resulted in low readings. In order t dependable values might be obtained, it was cessary that the entire membrane surface of membrane of the glass electrode be in contact th the material the p_H of which was being termined.[7] There was little difference in the of the nasal mucosa whether the contact for reference electrode was on the floor of the se near the point of contact of the glass elec- de or on the lip of the subject, as suggested Fabricant. The p_H values were usually about of a unit lower when the reference electrode s connected to the lip.

RESULTS

In 173 determinations on 142 persons ranging age from 17 to 95 years, readings varying m p_H 6.6 to p_H 7.6 were obtained. Of these, , or 72.3 per cent, were between p_H 6.8 and 7.2, and 68, or 39.3 per cent, were between 7.0 and p_H 7.1. Only 13, or 7.5 per cent, of determinations were below p_H 6.8, and 35, or 9 per cent, were above p_H 7.2. It appears m this study that the normal p_H of the nasal cosa lies in the region of neutrality, or possibly ghtly on the alkaline side.

These results are summarized in the table. In is are also given the p_H values of the mucosa of 20 persons with acute rhinitis. It is to be noted that the mean p_H value is essentially that of the normal nasal mucosa.

In one series in which the determinations were made on the same subject at intervals of five minutes for a period of seventy-five minutes, the variation recorded was between p_H 7.0 and 7.4. There was no correlation between the variation observed and the immersion of the subject's feet in cold water for a period of eight minutes. In another period of seventy-five minutes determinations were made of the p_H of the mucosa on each side of the nose at intervals of fifteen minutes. The variations in this period were from p_H 7.0 to 7.5. In another period of fifteen minutes, during which determinations were made at one minute intervals, the range was from p_H 7.1 to 7.4, and in a period of twenty-three minutes with determinations at intervals of one minute the variation was between 7.20 and 7.25. These readings were taken with the reference electrode connected by a bridge to the lip.

The p_H of Nasal Mucosa

	Age	Number	p_H Values		
			Low	High	Mean
Normal					
Men	17-30	85	6.6	7.5	7.06
Women	18-30	42	6.6	7.6	7.14
Men	60-95	15	6.7	7.5	6.98
With colds	.....	20	6.6	7.5	7.01

SUMMARY

It has been determined that the p_H of the nasal mucosa measured in situ in normal persons as well as in those with acute rhinitis is approximately 7.0. The slight variations from a neutral reaction which were observed were not considered sufficient to constitute an important factor in resistance to infectious diseases.

7. Kahler, N., and DeEds, F.: J. Am. Chem. Soc. :2998, 1931. Voegtlin, C.; Kahler, H., and Fitch, H.: Experimental Studies on Cancer: II. The Estiation of the Hydrogen Ion Concentration of Tissues Living Animals by Means of the Capillary Glass ctrode, National Institute of Health Bulletin 164, ited States Treasury Department, Public Health rvice, 1935, p. 15.

Case Reports

RETROPHARYNGEAL ABSCESS

Problems in Treatment

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Retropharyngeal abscess always causes much suffering of the patient and usually results in anxious moments for the family and the doctor. Moreover, retroesophageal and mediastinal extension probably occurs more often than is realized. It is always highly desirable, when possible, to determine whether such extension has occurred by means of lateral roentgenograms of the neck.

Retropharyngeal abscess is not especially common, nor is it uncommon. In hospital and private practice in the past fifteen years we have dealt with 27 patients showing this lesion. Thirteen of these patients were between 6 months and 5 years of age. Only 6 were adults (between the ages of 17 and 65). The remainder were children between 5 and 15 years of age. Sixteen of the 27 patients were female. The mortality was 3.7 per cent; that is, only 1 patient of the 27 died. Mediastinitis caused the death.

The dramatic impact of a recent case of retropharyngeal abscess impelled the making of this report because of the number and the complexity of the problems requiring solution.

A white housewife aged 32 was seen in emergency consultation at a suburban hospital because of sore throat, mucus accumulating in the throat and difficulty in speaking. She had been in the hospital overnight and had been well until a week before admission, when her throat had been raw and dry for four or five days. Then she felt almost well until the day she came to the hospital, when at 2 a. m. her neck felt stiff and she had severe knifelike pain in the throat. At 3 a. m. her neck started to swell, especially on the right side. Her throat felt full and "as if she had a stick running down behind her breast bone." Her voice was gradually reduced to a whisper.

She had been given irrigations of the throat and other local treatment together with administration of 16 Gm. of sulfadiazine (5 Gm. intravenously, 11 Gm. by mouth) in thirty-two hours. The twenty-four hour fluid intake had been 2,600 cc.; the output of urine, 360 cc. The patient had to strain to void. No gross hematuria had been observed. The sediment of the urine was normal on admission but showed 15 to 16 red blood cells per high power field twenty-four hours later.

The physical examination demonstrated well marked edematous swelling of all triangles of the right side of the neck with tenderness; less swelling and tenderness were present on the left side of the neck. In the edematous swelling were enlarged cervical lymph nodes.

The posterior pharyngeal wall, especially in the hypopharynx, seemed pushed forward. The right piriform sinus was obliterated. The epiglottis and the right arytenoid were somewhat edematous, and there was slight edema of the left arytenoid. However, there was no obstruction at the glottis. A red blush was present over the entire oropharynx and hypopharynx with edema of the right oropharyngeal and hypopharyngeal lateral wall, including the right tonsillar fossa. (The patient had had a clean tonsillectomy many years before.)

The temperature was 103 F.; the pulse rate, 140; the respiratory rate, 25; there was no cyanosis or laborec breathing.

The white blood cell count was 11,900, with polymorphonuclear leukocytes 84 per cent, small lymphocytes 4 per cent, large lymphocytes 10 per cent and eosinophils 2 per cent.

The patient was transferred to the Massachusetts General Hospital immediately.

A lateral roentgenogram of the neck was taken (*A* is figure), and the findings were reported by Dr. A. S. Macmillan as follows: "Marked retropharyngeal and retroesophageal swelling are seen, with the swelling extending into the superior mediastinum. The lung fields are clear."

In view of the oliguria and hematuria the problem requiring immediate decision was whether or not to continue the sulfadiazine therapy. The patient was given an intravenous clysis containing aqueous sodium racemic lactate 0.5 per cent concentration *and the treatment with adequate doses of sulfadiazine was continued.* Hot irrigations of the throat and applications of hot flaxseed poultices to the neck were ordered. A tracheotomy kit was kept at the bedside.

Within a few hours polyuria began, the output amounting to 3,600 cc. in twenty-four hours. The red cells disappeared from the urine. A high intake and output of fluids was maintained, and the urinary reaction was kept alkaline (*p*H value above 7). The white blood cell count was 21,200.

The temperature dropped to normal on the third day after the transfer to the Massachusetts General Hospital, and the cervical edema gradually subsided although prevertebral tenderness persisted. There was no respiratory embarrassment. The edema of the epiglottis and the arytenoids disappeared, but the displacement forward of the posterior pharyngeal wall continued and was most marked just superior to the arytenoids. The urine at this time was cloudy; the reaction, alkaline; the specific gravity, 1.014; albumin 1 plus; sugar, 0; diacetic acid, 0. The sediment contained 2 white blood cells and 30 epithelial cells. The white blood cell count was 11,700 with a differential count of 82 per cent polymorphonuclear leukocytes, 11 per cent small lymphocytes and 7 per cent large mononuclear leukocytes. The hemoglobin was 13.0 Gm. per hundred cubic centimeters.

Another lateral roentgenogram of the cervical region was taken on the fourth day after the transfer to the Massachusetts General Hospital.

A comparison of the actual size of the retropharyngeal swelling seen in this film with that in the previous one demonstrated only a very slight diminution in the diameters of the inflammatory mass, and it was decided to drain the abscess while the patient's condition was satisfactory and while the treatment with the sulfon-

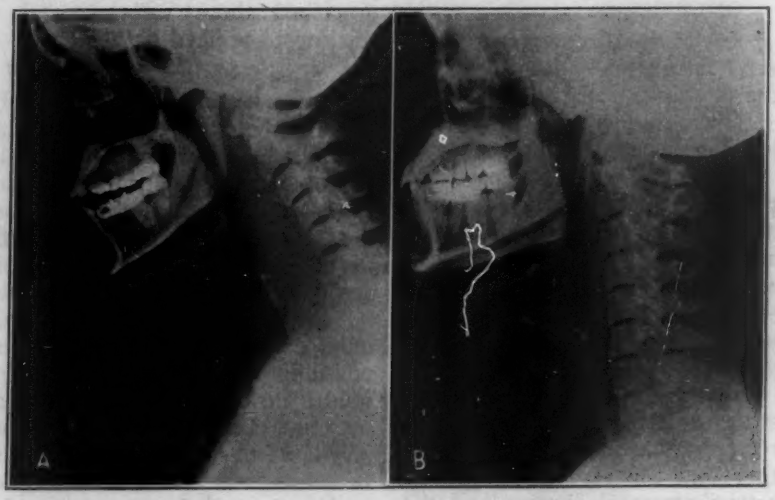
mide compound could be continued for a short time iterward without undue risk of further complications. The report on the last roentgen film had carried the emark: "The point of maximum swelling is just ehind the thyroid cartilage."

Was it good judgment to incise transorally such a arge abscess near the point of greatest bulging in a atient who had had laryngeal edema? Would this recipitate further edema? And would the wound stay pen sufficiently to drain an inflammatory swelling nown to extend into the superior mediastinum?

In spite of the desirability of not making a conpicuous scar in the neck of an attractive young woman, external incision and drainage of the involved region nder local anesthesia were advised. The advantage of lacing a drain properly and keeping it in situ as ong as necessary outweighed any minor cosmetic conideration. Moreover, while it was well known and idely accepted that treatment with a sulfonamide ompound would not affect undrained pus, the effect on *he walls of the abscess,* especially one of this size, *ould not be predicted with certainty.* This fact was orne out by the operative findings.

gentle retraction of the carotid sheath brought the capsule of the abscess into view. This was extremely tender, and it was impossible to proceed further under the local anesthetic, which was then supplemented with pentothal sodium intravenously injected. A total dose of 0.5 Gm. was used during the remainder of the operation. It was given slowly and intermittently.

The anesthetist warned the operator *before* starting the injection of pentothal sodium that the *respiration would stop momentarily from laryngeal spasm (not* from failure of the respiratory center). This occurred almost simultaneously with the loss of consciousness after a fraction of the drug had been slowly, intermittently and cautiously injected. This raised the question: Why not insert a bronchoscope? This would merely have made the spasm worse and might have induced vomiting (which is not abolished by pentothal) with disastrous results, possibly causing the capsule of the abscess to rupture. Artificial respiration was avoided, also. After a fraction of a minute breathing started spontaneously. *No surgical manipulation of any kind was done for three or four minutes,* in order to avoid possible recurrence of further laryngeal spasm.



A, lateral roentgenogram of the neck showing marked retropharyngeal and retroesophageal swelling.
B, lateral roentgenogram of the neck showing regression to normal.

The risks of inducing general anesthesia in a patient with retropharyngeal abscess are so great as to rule out such a procedure if it is humanely possible to avoid it. Local anesthesia is highly preferable for the external approach and *no* anesthesia for the transoral operation.

When the decision to operate was made in this case, no general anesthetic could be selected whose disadvantages did not seem paramount as compared with the advantages of procaine hydrochloride, especially as in meeting previous similar problems local anesthesia had seemed to work well.

However, it was realized that manipulation around the capsule of the abscess might cause it to rupture and discharge its content into the pharynx; therefore suction apparatus, together with a laryngoscope and a bronchoscope, was set up in the operating room before proceeding with the operation. Then, with the field of operation under local anesthesia and with no other preoperative preparation than the subcutaneous injection of 1/150 grain (0.43 mg.) of atropine sulfate, an incision was made at the posterior border of the right carotid triangle, exposing the anterior margin of the sternomastoid muscle. Dissection of the cervical fascia and (The technic of inducing anesthesia with pentothal sodium has been well reported by Thomas.[1])

Then an entrance into the abscess cavity was made by blunt dissection. Approximately 12 cc. of yellow-gray pus was obtained. This was surprising because of the size of the abscess and because with similar patients *not* treated with sulfonamide compounds huge quantities of pus had been drained. Finger exploration of the abscess cavity revealed a striking mass of granulations everywhere on the walls, making this layer many times thicker than the ordinary abscess wall. A Miller tube drain was placed in the cavity, and the external wound was partly closed.

The patient's further convalescence was completely uneventful. The administration of the sulfonamide compound was stopped on the seventh postoperative day. The drain was gradually withdrawn and was out by the sixth postoperative day. The patient was discharged to her family doctor on the eighth day after

1. Thomas, G. J.: Pentothal Sodium in Ear, Nose and Throat Surgery, Ann. Otol., Rhin. & Laryng. **52**:35 (March) 1943.

operation with a draining fistula, which healed in approximately a week.

The findings in a check-up roentgenogram of the neck (*B* in the figure) were reported by Dr. A. S. Macmillan two weeks after the operation as follows: "The retropharyngeal structures are perfectly normal, and the swelling of the mediastinum has disappeared."

Richards [2] listed common complaints in 162 cases of retropharyngeal abscess as dyspnea (61 cases), dysphagia (59 cases), swollen glands (37 cases), stiffness in neck (31 cases), fever (27 cases), sore throat (9 cases), cough (3 cases) and convulsions (2 cases). He laid great stress on failure to suspect the possibility of a retropharyngeal abscess.

Grodinsky [3] emphasized that extension of retropharyngeal abscess to the superior or the posterior mediastinum is indicated by pain in the chest, dyspnea, persistence or recurrence of fever and roentgen evidence of mediastinitis.

COMMENT

The anatomic spaces of the pharyngomaxillary fossae have been adequately described by Mosher [4] and others. Since Mosher's practical and classic description there has been a tendency to split the cervical fascia between the prevertebral layer and that covering the constrictor muscles of the pharynx into more or less academic subdivisions. Weintraub [5] indicated the possibility anatomically of sepsis occurring between the constrictor muscle fibers themselves and their fascial covering—the "parapharyngeal space." Iglauer [6] expressed strong belief that the so-called retropharyngeal abscess actually begins in most cases as a peripharyngeal abscess with infection in the space described by Weintraub [5] and not in the retropharyngeal space itself. He pointed out further that the clinical site of the more common retropharyngeal abscess and the anatomic site of the peripharyngeal space coincide and that a simple retropharyngeal abscess results in breaking down of the infected lateral retropharyngeal lymph gland or glands, which in turn have an intimate anatomic relationship with the upper portion of Weintraub's [5] parapharyngeal space. In spite of this painstaking research we are impressed by the frequency of the large retropharyngeal inflammatory swelling as demonstrated by roentgen examination in our cases.

The truth probably is that there is a remarkable ability of the cervical fascia to produce walling off in the presence of any inflammatory stimulus. This fact has been utilized by Lahey [7] in his two stage operation for pulsion diverticulum of the esophagus in which a cigaret drain is utilized to secure walling off of the operative field from the superior mediastinum and lower part of the neck after the first stage is done.

From the point of view of pathology little comment need be made, since ordinary principles of inflammation are borne out when applied to the anatomic spaces of the pharyngomaxillary fossae. It is widely accepted that lymphadenitis of the retropharyngeal gland, induced by inflammation in the nose, the pharynx or occasionally the ears, with subsequent breaking down of the gland, is fundamentally important. In the cases described there were several striking points relating to applied pathology:

1. Easily observable edema of the larynx was present without respiratory embarrassment. It is likely that such edema of the larynx is present more frequently than is commonly supposed, but because of the difficulty of indirect laryngeal examination in this type of case, especially if the patient is a small child, and the risk of spontaneous rupture of the capsule of the abscess on any such manipulation as direct laryngoscopy, no satisfactory laryngeal examination is done unless respiratory embarrassment occurs. The possibility arises that death from respiratory failure in some cases of retropharyngeal abscess may have been associated with partial edematous laryngeal obstruction. In any event, while conservative treatment of the retropharyngeal abscess is being carried out, means for dealing with laryngeal obstruction should be available at the bedside and certainly in the operating room when any form of surgical treatment is done.

2. A second observation of much interest was the thick wall of the abscess cavity, which gave the impression of an attempt by nature toward wide organization of the greater portion of the inflammatory swelling, failing only because *sulfonamide compounds will not affect undrained pus.* In a similar case the transoral incision might offer less certainty of finding and draining a relatively moderate amount of pus in what seems by roentgenogram to be an enormous easily drained abscess.

3. The wide extent of retropharyngeal inflammation makes the taking of fluids by mouth difficult, especially if retroesophageal and mediastinal swelling are present; but it is axiomatic in the administration of sulfonamide compounds that the intake of fluids be kept at adequate

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evels (and with the use of sulfadiazine that the rinary reaction be kept alkaline [p_H above 7]). Therefore, in cases of retropharyngeal abscess is imperative that the oral intake of fluids be upplemented parenterally if necessary. The best eans of judging whether the intake of fluids is dequate is to make certain that the urinary out- ut measures at least 1,000 cc. or better about ,500 cc. daily. Since sulfadiazine is more oluble in an alkaline than in an acid medium, a urse should determine the urinary p_H by means f nitrazine paper several times daily and in- rease the intake of alkali if the p_H is below 7. lkali is most easily administered intravenously s aqueous solution of racemic sodium lactate.

4. The fact that retropharyngeal, retroeso- hageal and mediastinal swelling (*A* in figure) ay subside uneventfully and completely (*B* in gure) with adequate drainage and administra- on of a sulfonamide compound is illustrated y this report. The great value clinically of teral roentgenograms of the neck to show the extent and the degree of swelling in pharyngo-maxillary inflammation cannot be questioned.

5. If a large retropharyngeal abscess or retropharyngeal-retroesophageal abscess is demonstrated by roentgen examination and if a sulfonamide compound has been administered in adequate dosage for several days, it must not be assumed that the abnormal mass consists mostly of pus, as was logical before the advent of sulfonamide drugs. A large portion of the swelling may consist of organizing granulations. *In such a case transoral drainage is relatively safe in that there is little danger of the patient aspirating pus.* Yet under such conditions there is greater difficulty in striking the pus when an incision is made transorally. While this type of surgical approach is undoubtedly adequate in the majority of cases of retropharyngeal abscess, external drainage has a definite and valuable place in the treatment of such patients as the one whose case is reported.

395 Commonwealth Avenue.

Clinical Notes; New Instruments and Technics

RETAINING A CORRECT SEPTOLABIAL ANGLE IN RHINOPLASTY

JACOB DALEY, M.D., NEW YORK

For many years rhinoplastic surgeons have been confronted with the problem of preventing a widening or a weblike distortion of the septolabial angle following the classic procedure for shortening the nose, i. e. (1) transsection of the membranous septum down to the anterior nasal spine of the maxilla, (2) reduction of the septum and upper lateral cartilages and (3) reattachment of the columella to the septum.

In carrying out step 3 overcorrection is essential to compensate for the subsequent inevitable contraction due to cicatrization of the transfixed membranous septum. With this in mind, Jacques Joseph, of Berlin, in 1898 devised the septocolumellar suture to raise the tip of the nose,[1] and this method became the standard practice in rhinoplasty and is meticulously followed at the present time. A heavy silk suture is passed through the septal cartilage and the columella in an oblique fashion, so that when the suture is tied the mobile tip of the nose is projected forward and upward, the fixed septum acting as a fulcrum (fig. 1 *A* and *B*). While this procedure accomplished the desired purpose, it nevertheless resulted in a distorted septolabial angle (fig. 5 *A* and *B*), the amount of distortion being in direct proportion to the degree of elevation of the nasal tip.

It occurred to us of the Fomon Group that a restraining suture introduced at the angle would obviate such distortion and at the same time permit the required amount of elevation. Of the restraining sutures, one may choose from the following:

1. Joseph's[1] suture. A heavy silk suture is passed through the dorsocaudal margin of the septal cartilage and tied over the columella so that the lip is recessed to maintain a correct septolabial angle (fig. 1 *C*). This suture was necessarily under considerable tension and herein lay a great objection. Not infrequently the suture cut through the septal cartilage or caused necrosis of the soft tissue.

2. Kazanjian, V. H., cited by Fomon, S.: Surgery tape strapped firmly across the nasolabial angle. While it is true that with this method there is no danger of necrosis, in my hands it was found to be entirely unsatisfactory.

3. Gorney[3] devised an ingenious method. He reasoned that since considerable tension is necessary, the bony

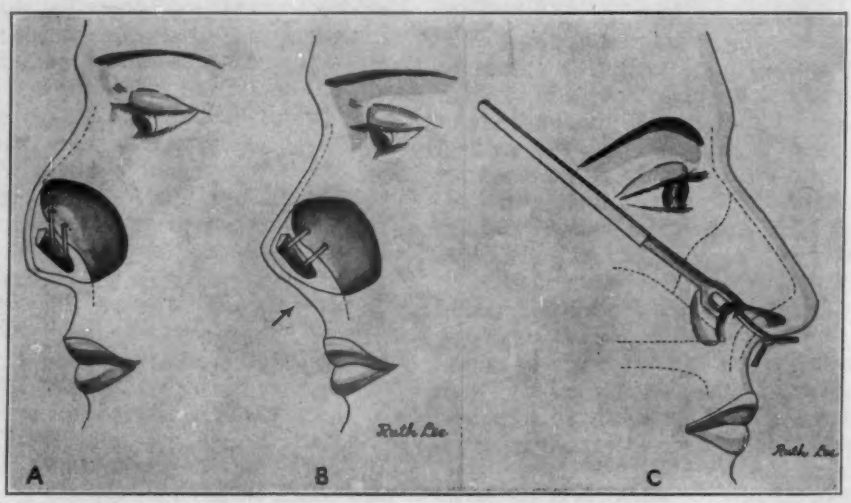

Fig. 1.—Orthopedic suture of Joseph[1]: *A*, suture passed through the septal cartilage and the columella in an oblique fashion. *B*, suture tied. The arrow shows widening of the septolabial angle after elevation of the tip. *C*, suture to maintain a correct septolabial angle (Joseph[1]). Note pressure on the skin and tissues at the nasolabial angle.

Read at the scientific meeting of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery held Feb. 25, 1944, at the Academy of Medicine, New York.

1. Joseph, J.: Nasenplastik und sonstige Gesichtsplastik, Leipzig, Curt Kabitzsch, 1931.

2. Kazanjian, V. H., cited by Fomon, S.: Surgery of Injury and Plastic Repair, Baltimore, William Wood & Company, 1939, pp. 731-732.

3. Gorney, A.: Personal communication to the author.

argins of the piriform opening might well be used as nchor points for the ligatures. Two heavy sutures were assed through holes drilled through the piriform marins on each side. These two anchored sutures were en tied over the well padded nasolabial angle. The

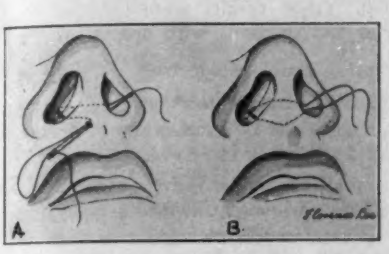

Fig. 2.—Submerged perforating suture: *A*, needle ntroduced through the dorsocaudal end of the septal artilage and passed through the columellar skin. The ourse is retraced through the perforation of the skin n the columella. *B*, suture passes out through opposite ostril ready for tying.

ationale and the basic principle of this method are xcellent inasmuch as considerable tension can be aplied with safety, and the technic is not too difficult. Yet t seems to me that the procedure is far too cumbersome o warrant its use as a routine measure.

4. Aufricht[4] recently described an interesting proedure in which a submerged perforating suture is so assed that the upper lip is set back in the proper osition. The needle is introduced through the dorsoaudal end of the septal cartilage and made to pass

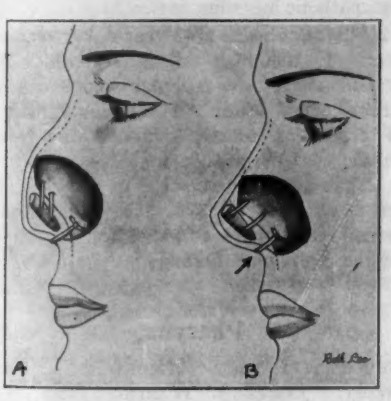

Fig. 3.—Combined orthopedic and submerged perforating sutures: *A*, submerged perforating suture tied and orthopedic suture in position. *B*, orthopedic suture tied. The arrow shows the correct nasolabial angle after elevation of the tip.

etween the mesial crura and out through the columellar kin. The course of the suture is then retraced through the perforation in the skin of the columella, back again between the mesial crura, out through the opposite nostril (fig. 2), and tied. The external loop disappears in the skin puncture but it catches enough subcutaneous tissue to hold the columella and the lip firmly in the proper position and so obliterates the wide septolabial angle.

It became obvious that the Joseph orthopedic suture used in combination with a good restraining suture would solve our problem. We have been using such a combined suture routinely and find it most satisfactory. We no longer fear the marring of a well executed rhinoplasty by a septolabial distortion.

In brief, the technic is as follows: Following a "typical rhinoplasty" the columella is fixed to the dorsocaudal end of the septal cartilage by the use of the "submerged perforating suture" as described (fig. 2). This is followed by the use of the conventional ortho-

Fig. 4.—At left, the conventional orthopedic suture used alone: *A* and *B*, before and after. Note the widened nasolabial angle. At right, the conventional orthopedic suture used in conjunction with the submerged perforating suture: *C* and *D*, before and after. Note the correct nasolabial angle.

pedic suture (fig. 1 *A* and *B*). The joint use of both sutures (fig. 3) gives excellent results and eliminates an annoying episode in corrective rhinoplasty.

A comparison of the photographs showing Joseph's orthopedic suture alone (fig. 4 *A* and *B*) and those showing the combined orthopedic and submerged perforating sutures (fig. 4 *C* and *D*) tells the story much better than words.

I have found this suture to be the only procedure that will accomplish the purpose. The idea is so simple that it is hard to believe that others have not thought of it and in all probability it has occurred to many others. The procedure has been so gratifying in our hands that we suggest its wider use.

104 East Fortieth Street.

4. Aufricht, G.: A Few Hints and Surgical Details in Rhinoplasty, Laryngoscope **53**:331 (May) 1943.

Abstracts from Current Literature

Ear

SOUND TRANSMISSION THROUGH THE EAR AND ITS RELATION TO SOUND INJURY. FREDERICK M. GROSSMAN, Ann. Otol., Rhin. & Laryng. **52**:666 (Sept.) 1943.

The initial symptom of injury to the ear by sound is a "tonal dip" in the frequency scale, which appears most frequently at 4096. The author has made a survey of the data concerning quantity of sound transmission as related to sound perception of normal ears under normal conditions and under sound overload. He summarizes his findings as follows: 1. A loud noise effects a contraction of the tensor tympani muscle reflexively, which may persist long after the cessation of the injurious noise. 2. In normal ears, with contraction of the tensor tympani muscle the following adjustments are noted: Sound transmission is reduced at low as well as at high frequencies, with one minimum in the higher middle range; the natural frequency of the vibratory system in the middle ear is increased. There is reason to assume that the impedance of the drum is changed in such a manner that the resonance peaks in the external ear canal are sharper and higher than under normal conditions.

Grossman feels that the natural frequency of the middle ear mechanism approaches that of the region of resonance in the external auditory canal, thus facilitating materially the transmission of frequencies in the neighborhood of 2,000 cycles per second relative to other frequencies of the sound spectrum. It seems probable that the tonal dip is caused by injuring intensities in that part of the tonal spectrum which is least protected by the reflex action of the middle ear against injurious noise. The resulting damage is a combination of the action of resonance in the external canal and resonance in the middle ear.

M. V. MILLER, Philadelphia.

MÉNIÈRE'S VERTIGO. A. ASENJO G. and J. S. RIESCO, Rev. de otorrinolaring. **2**:83 (Dec.) 1942.

Asenjo and Riesco discuss vertigo and classify the symptoms as: 1. Ménière's disease, which is due to a hemorrhage in the labyrinth and is associated with deafness and vertigo. 2. Ménière's syndrome, which presents the same triad of symptoms but which in other characteristics, in etiology and in paroxysmal occurrences varies considerably from Ménière's disease. 3. Pseudo Ménière's disease, in which the clinical picture is incomplete. 4. A syndrome in which the symptoms of Ménière's disease are definitely associated with medical disorders, with neurologic or neurosurgical conditions, with intracranial tumors and with vascular lesions (aneurysms).

Stress is laid on the differential diagnosis between these four groups. An important aid is the vestibular examination, which detects an intracranial tumor.

The treatment is both medical and surgical. The former must be tried first. Often the symptoms disappear spontaneously. The authors follow the Furstenberg routine, emphasizing either ammonium chloride or potassium nitrate as diuretics, with the addition of low intake of fluids. Vitamin B in large doses is also advocated. Potassium chloride has been used in several cases with good results.

The surgical treatment is section of the vestibular portion of the eighth nerve as advocated by Dandy.

The authors have a series of 25 cases, 18 medical and 3 surgical. Good results were obtained in 12 of the first series and in the 3 with operations.

PERSKY, Philadelphia.

FUNCTIONAL EXAMINATION OF THE EIGHTH NERVE. J. S. RIESCO and C. FERNANDEZ F., Rev. de otorrinolaring. **2**:183 (March) 1943.

Riesco and Fernandez have made a series of 33 functional tests in patients with all types of intracranial lesions. These lesions included tumors in all sites, injuries of skull and brain, cerebral hemorrhage, intracranial hypertension, jacksonian and idiopathic types of epilepsy, aneurysms, arteriosclerosis and various types of vertigo.

The authors present 2 otologic cases which illustrate some of the difficulties encountered in the interpretation of the cochlear-cerebellar-vestibular tests.

CASE 1.—A man 50 years old complained of left trigeminal neuralgia of five years' duration. Recently this was complicated by marked tinnitus and deafness. In addition there were mydriasis of the left pupil and exophthalmos. In view of the left-sided deafness, spontaneous nystagmus to the right and peripheral palsy due to disease of the facial nerve on the left side with slight involvement of the left glossopharyngeal and hypoglossal nerves, the otologist made a diagnosis of tumor of the left cerebellopontile angle. A ventriculogram and subsequent operation failed to reveal any tumor mass. Subsequently the patient improved under treatment with ergotamine tartrate so that the final diagnosis was trigeminal neuralgia due to hyperfunction of the sympathetic nervous system.

CASE 2.—Symptoms in a 42 year old woman indicated involvement of the fifth, sixth, seventh, eighth and ninth cranial nerves. Vestibular and neuralgic signs pointed to a central lesion. The otologist made a diagnosis of cerebellar tumor, but operation revealed a tumor in the cerebellopontile angle.

These 2 cases exhibited some of the difficulties encountered in arriving at a correct diagnosis and stress the responsibility of the otologist for proper interpretation of the vestibular findings.

PERSKY, Philadelphia.

Pharynx

BACTEROIDES SEPTICEMIA: REPORT OF A CASE WITH RECOVERY. JAMES J. WARING, JOHN G. RYAN, RICHARD THOMPSON and FRANK R. SPENCER, Laryngoscope **53**:717 (Nov.) 1943.

Septicemia considered as having origin in streptococcic pharyngitis or tonsillitis may in reality be Bacteroides septicemia. Bacteroides is a genus of gram-negative organisms, which may or may not be pathogenic. They are anaerobic.

The clinical picture is that of tonsillitis or pharyngitis, at first mild, then becoming severe, with chills and high fever occurring by the fourth day or after. There may be complications, such as peritonsillar abscess or extension into the jugular vein. Occasionally there is pneumonia with pleurisy or septic arthritis. Metastatic abscesses may occur.

A case is presented, with recovery, probably due to eatment with sulfonamide compounds. A culture of aterial from the throat showed hemolytic streptoci, and the infection was so diagnosed in the beinning. Blood cultures, however, showed Bacteroides two occasions. Several complications occurred.

The authors present the bacteriologic properties of e organisms in this case and the methods used in owing and identifying them. They were found to long to the species Bacteroides funduliformis.

HITSCHLER, Philadelphia.

ERILARYNGEAL CYSTS. JUSTO M. ALONSO and ELIAS REGULES, Rev. de otorrinolaring. **3**:15 (June) 1943.

Alonso and Regules present 2 cases of perilaryngeal sts. These are congenital laryngeal cysts or cystic ryngoceles. They are found on the inner and the per part of the thyroid cartilage and outside of the ryngeal muscles. The principal finding is an elevaon of the base of the epiglottis and the ventricular nd. Both patients were treated surgically. The cysts ere removed by an external incision, and there were recurrences.

PERSKY, Philadelphia.

Nose

BSERVATIONS ON PARANASAL SINUSITIS AMONG PATIENTS SUBJECTED TO LOBECTOMY FOR BRONCHIECTASIS. KINSEY M. SIMONTON, Ann. Otol., Rhin. & Laryng. **52**:598 (Sept.) 1943.

Simonton reviews much of the literature regarding e mechanics of the spread of infection between the per and the lower respiratory tract. He reviewed a ries of 405 consecutive cases in which chronic sinusitis as diagnosed at the Mayo Clinic and noted the incince of bronchiectasis. Only cases in which the history dicated the presence of sinusitis for longer than one ar were considered. Symptoms of sinusitis had been resent for one to five years in 212 cases, for five to n years in 60 cases and for more than ten years in 3 cases. Bronchiectasis was recorded in 23 (5.7 per nt) of the 405 cases. In 11 of these the onset of nusitis and that of bronchiectasis were simultaneous. n no case was there a clear history of bronchiectasis receding sinusitis. In 11 of the 23 cases it seemed finite that the onset of bronchiectasis followed pneuonia or the acute diseases of childhood.

The author has observed that the results of treatment sinusitis in the presence of bronchiectasis are poor. he early postoperative course is one of considerable provement in the condition of the sinuses with a finite decrease of, or complete relief from, purulent scharge in the nose. After the cessation of postperative treatment, however, the tendency is toward gradual return of purulent nasal discharge in a large roportion of cases, accompanied by further pathologic anges in the sinus area. This is the result of repeated scharge of infected secretions from the lungs into the se during coughing. He feels that in certain patients ith bronchiectasis, young people and those in whom e disease is limited to a small portion of the lung, e condition can be successfully treated by surgical moval of the diseased portion of the lung. Following is procedure there is immediate cessation or marked crease of productive cough, dependent on whether l of the bronchiectasis was eliminated by operation d on the degree of chronic bronchitis present in the maining lung. After lobectomy the prospect of sucsful treatment of sinusitis is improved to a degree proportional to the productive cough. He gives several case histories to support this.

M. V. MILLER, Philadelphia.

OSTEOMA OF THE FRONTAL SINUS. R. DE VILLIERS, Vida nueva **51**:125 (April) 1943.

Villiers reports a case of osteoma of the frontal sinus in which the only symptoms were spontaneous pain and tenderness to pressure along the superciliary ridge. This pain was unilateral, and after extirpation of the tumor there were no recurrences of either the symptoms or the tumor.

PERSKY, Philadelphia.

Miscellaneous

TONSILLECTOMY AND ACUTE NEPHRITIS. JOHN B. LYTTLE, Bull. New York Acad. Med. **18**:356 (May) 1942.

Although the causation of acute nephritis is variable both in the nature of the causative organism and in the specific damage done to the kidney, the author feels that since the throat is the most frequent portal of entry for pathogenic agents any procedure which tends to diminish the occurrence of sore throat may be considered a potential block to the source of danger. For this reason, whenever indications for tonsillectomy exist, the tonsils should be removed as a precautionary measure against the development of nephritis.

Nephritis itself, the author states, is not to be considered an indication for tonsillectomy. This is an elective operation, and the time must be decided with regard to the individual case; it preferably is when the symptoms and the signs of the acute stage of nephritis have subsided and the pharynx is not acutely inflamed.

JAHR, Omaha. [AM. J. DIS. CHILD.]

FUNCTIONAL SPEECH AND VOICE DISORDERS. JAMES S. GREENE, J. Nerv. & Ment. Dis. **95**:299 (March) 1942.

Stuttering is a symptom the genesis of which is in the emotional stream rather than in the vocal structures, and treatment, to be successful, must be directed toward the whole personality pattern. Reeducation of muscles of speech, however, is at times an important phase of therapy. The whole problem of stuttering is comprehensively discussed. The author bases his ideas and conclusions on personal experiences in over 20,000 cases.

JAHR, Omaha. [AM. J. DIS. CHILD.]

THE ETIOLOGY OF STAMMERING. ELMER L. KENYON, J. Speech Disorders **7**:97 (Aug.) 1942.

Vocal cord adduction is a life-saving function. Voice production is a modification of adduction. In developing mental imagery for control of the vocal cords, a child may, under stress of confusion, mental excitement or emotional strain, develop an imagery of complete cord adduction as being the way to produce voice. Thus a complete but transient block develops on phonation, which is the fundamental symptom of "stammering." Other symptoms are caused by social pressures of ridicule and embarrassment on the child. The author considers his theory proved by the pragmatic test of no failures under a treatment designed to draw the child's attention to proper vocal production. For more than seven years of work, he had no failures with children between the ages of 8 and 12 years. The author states that these are the most favorable ages for the program of treatment that he designed. Manner of treatment,

number of cases and methods of control are not discussed by the author in this article.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

REPORT OF A SPEECH SURVEY, HOLYOKE, MASSACHUSETTS. ALICE W. MILLS and HELEN STREIT, J. Speech Disorders **7**:161 (Aug.) 1942.

All of the children in the first three grades were examined by ten examiners, not all of them trained in speech correction. Above the third grade, children were seen who were referred by teachers as being defective in speech. Of 4,685 enrolled in the schools, 10.1 per cent were found to have some defect of speech. While on the average there was a sharp reduction in the total number of defects above the third grade (33 per cent vs. 10.1 per cent), there was a large increase in the number of serious ones in ratio to the total number of defects. A number of other statistical conclusions are of interest. The sex index of speech defects is again confirmed, the greatest variant being in stuttering, in which 5 boys to 1 girl stuttered.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

STUDIES IN THE PSYCHOLOGY OF STUTTERING: XVII. A STUDY OF THE TRANSFER OF THE ADAPTATION EFFECT IN STUTTERING. WILLIAM E. HARRIS, J. Speech Disorders **7**:209 (Sept.) 1942.

Twenty stutterers read a 180 word passage four times, a similar 180 word passage fifteen times and another 180 word passage four times. Stuttering is reduced as a stutterer rereads the same passage. This reduction apparently also occurs when he reads a new passage similar to the one he has just read several times. This adaptation effect does not extend to conversation.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

A WORKING HYPOTHESIS AS TO THE NATURE AND TREATMENT OF STUTTERING. JULIAN B. ROTTER, J. Speech Disorders **7**:263 (Sept.) 1942.

The early history and a psychologic method of treatment of 8 stutterers are presented. No speech training of any kind was given the stutterers, but a therapy was worked out on the basis that stuttering is a defense mechanism. In 2 cases the condition was completely corrected, and in 2 it was nearly corrected; in the others it was unimproved. The author feels that stuttering is a result of a sensation of inadequacy which is produced entirely exogenously, that pampering is commonly found in the early histories of young stutterers and that the subject's attitude toward stuttering is characteristic of his whole attitude toward life.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

OSTEOCHONDROMAS ARISING FROM THE BASE OF THE SKULL. CARL FELIX LIST, Surg., Gynec. & Obst. **76**:480, 1943.

List reports 7 cases of osteochondroma of the skull, the tumor being primarily intracranial, arising from the sphenoid bone, in 5 and primarily extracranial, arising from the ethmoid or sphenoid region, with secondary intracranial extension in 2. In each case of primary intracranial osteochondroma the growth originated from the sphenoid bone and extended intracranially into the posterior parasellar region. In 1 case the growth was but the intracranial manifestation of generalized chondromatosis. The characteristic site of osteochondroma makes probable that it develops from a residual of the cartilaginous primordial cranium. The author notes that chordoma is found in a location similar to that of osteochondroma, viz., at the junction of the basisphenoid and the basiocciput.

Osteochondroma is usually seen in young adults and grows slowly; hence a course of over ten to twenty years is not unusual. Intracranial osteochondroma produces a neurologic syndrome characteristic of its parasellar location. Osteochondroma that is primarily extracranial produces symptoms at first characteristic of an expansive or obstructive lesion of the paranasal sinuses. Later, as the tumor penetrates the cranial cavity, the optic nerve is first involved, and later a parasellar syndrome may be produced. Roentgenograms are almost pathognomonic.

The treatment is surgical. The prognosis is good provided cancerous change has not occurred. Recurrence ultimately results, however, since only partial removal is possible.

SHENKIN, Philadelphia.
[ARCH. NEUROL. & PSYCHIAT.]

ALLERGY FROM THE STANDPOINT OF OTORHINOLARYNGOLOGY. MARIO RIUS, An. de oto-rino-laring. Uruguay **12**:149, 1942.

In discussing the difficulties encountered in establishing a correct diagnosis of allergy, Rius emphasizes the importance of eosinophilia as observed in routine nasal smears and differential blood counts. The incidence of eosinophilia should be determined routinely and added to the data from the usual complete laboratory and roentgen studies.

He mentions the many allergic manifestations which may occur in the ear, the nose, the throat, the larynx or the eosophagus. These manifestations are usually extremely resistant to the ordinary routine therapeutic measures. In addition they present many atypical features which may cause uncertainty as to the correct diagnosis. However, once a diagnosis of allergy has been made, the causative factors must be determined. Therapy consists of the employment of elimination diets, desensitization to the specific allergens and administration of calcium gluconate, which may be given either orally or parenterally.

PERSKY, Philadelphia.

IONIZATION IN OTOLARYNGOLOGY. MARIO A. CASSINONI and BARTOLOME FUSTER, An. de oto-rino-laring. Uruguay **13**:1, 1943.

The authors discuss the role of ionization, stressing the number of different electrolytic solutions which they have used in various infections. The first solution was a 1 to 2 per cent solution of potassium iodide or calcium chloride. The treatment recommended was to give 5 to 10 milliamperes of a galvanic current for a period of approximately thirty minutes. This treatment should be repeated in series in which six daily treatments are given for a week, then three treatments a week for three weeks, totaling 15 applications. The authors usually wait three weeks after the last treatment before repeating the series. In addition they supplement the treatment by administering internally one hour before the treatment 1 Gm. of either potassium iodide or calcium chloride, depending on which electrolyte is employed in the treatment.

With respect to ionization of the ear, the following suggestions are made: 1. It is contraindicated in the presence of acute localized suppuration in the mastoid cells. 2. Optimum results are obtained only when the infection is localized to the tympanic cavity proper. 3. The external canal should be cleansed thoroughly

d small tympanic perforations should be enlarged. ny external otitis should be cleared up completely prior the treatment. 4. If there is evidence that the stachian tube acts as a source of reinfection, attention ould be directed to the pharynx and nasopharynx. denoid vegetations and infected tonsils should be re-oved, and dental caries and sinusitis should be cleared . 5. Aural polypi and granulations must be removed ior to treatment. 6. Infected contents of the attic may aspirated, but necrosis of bone, either in the attic or the mastoid, is a contraindication to treatment. 7. etrositis is always a contraindication. The authors sually employ a 1 per cent solution of zinc sulfate, iving 1 to 3 milliamperes for twenty to thirty minutes. o after-treatment is necessary.

They have employed this method of treatment in cer-in cases of tinnitus. In such cases, however, they e a 1 per cent solution of sodium silicate and give a eries of ten applications within two weeks, which ay be repeated after a two week interval.

They have employed this method of treatment for a umber of nasal conditions with varying results, using rincipally a 1 per cent solution of zinc sulfate as the lectrolyte. They have also tried this method of treat-ent in a series of cases of neuralgia of the face, ither trigeminal or glossopharyngeal. Among these ases were instances of the atypical facial neuralgia, of luder's syndrome, of Sharlin's neuralgia, of the sychalgia of Sicard and of glossopharyngeal neuralgia. n this series of cases of neuralgia they have employed 0.25 per cent solution of aconite nitrate as the electro-yte. In addition they recommend the internal admin-stration of a solution of equal parts of tincture of elladonna and alcoholic extract of aconite, twenty rops three times a day. The method of application is o apply the negative electrode to the neck in the egion of occipitovertebral articulation, and the positive lectrode moistened in the 0.25 per cent solution of conite nitrate to the root of the nerve or its painful istribution.

In their conclusions based on 38 cases of various ypes of neuralgia they observed that ionization with conite is a painless method without any danger and without any complications. There are no contraindica-ions, and in most cases there is total elimination of he pain. While it may not cure the initiating factors f the syndrome it acts as an auxiliary treatment and n of partial benefit without any risk. The action may e less rapid than that of an injection of alcohol or hat of electrocoagulation of the nerve, but it does not ave the dangers associated with the latter procedures.

PERSKY, Philadelphia.

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. ENRIQUE GRUNWALD S., Rev. de otorrinolaring. **2**:161 (March) 1943.

Grunwald in a comprehensive review of observations n thrombophlebitis of the cavernous sinus stated that he involvement usually originates from an infection in he skin of the nasal vestibule, in the maxilla, in an yelid or in the forehead. In its initial stage this in-fection appears as a small circumscribed abscess with imited, circumscribed thrombophlebitis of the surround-ng small veins. If at this time the furuncle is either ncised or traumatized, the infection may be disseminated hrough the small veins into the larger venous system, where it then may extend into the cavernous sinus and end fatally. Thus a small localized infection can be transformed into generalized septicemia.

The venous drainage into the cavernous sinus is usually effected by one of two routes: (1) the superior ophthalmic vein, which receives blood from the fore-head, the eyelids, the nose, the genionasal sulcus and the upper lip, and (2) the inferior ophthalmic vein, which drains the floor of the orbit, the maxilla, the inferior eyelids, the lacrimal sac, the nasolacrimal canal and the nasal fossae. These vessels transverse the orbit from before backward and unite to form the ophthalmic vein, which passes through the sphenoidal fissures and empties into the cavernous sinus.

There are three routes by which an infection can reach the cavernous sinus. First is the anterior route, which is by way of the ophthalmic vein as already described. A suppurative process of the frontal sinuses, a hematoma of the septum and other infections in the nasal chamber can follow this route and involve the cavernous sinus. Second is a lateral route by way of the veins of the foramen ovale. These receive blood from the pterygoid venous plexus. This plexus drains the buccal mucosa, the dental alveolus, the pharynx and the tonsils. Third is the posterior route by way of the superior and inferior petrosal sinuses. These sinuses have communication with the lateral sinus and the internal jugular vein, respectively. This relation between the cavernous sinus and the venous circulation of the temporal bone and also the superior part of the neck explains a number of cases of cavernous sinus thrombosis which took origin in mastoiditis or cervical adenitis.

The symptoms of thrombophlebitis are due to a sudden blockage of the return circulation with a secondary infiltration of the orbital tissues. The most important symptoms are palpebral edema, conjunctival ecchymosis and exophthalmos. These symptoms are usually bilateral. There is also ophthalmoplegia, edema of the lids and loss of vision. There is also evidence of generalized septicemia, such as pyrexia, chills and finally symptoms of complicating meningitis.

The prognosis is always extremely grave. Prior to the advent of sulfonamide compounds the mortality rate was 100 per cent. Death usually ensued four to ten days after the onset of symptoms. Now with the intensive use of the sulfonamide drugs, an occasional cure has been reported in the literature. Grunwald observed three cures and two deaths recently.

He summarizes the various surgical technics that have been advocated. These are all of doubtful value. He stresses the importance of prophylaxis.

In conclusion Grunwald outlines the therapy as essentially chemotherapy, blood transfusions, the use of serums and vaccines, lumbar punctures, the administration of cardiac stimulants and sedation.

A CASE OF STREPTOCOCCUS MENINGITIS IN A NURSING BABY, WITH RECOVERY. E. G. CASELLI, Rev. Soc. pediat. de la Plata **3**:81 (Jan.-April) 1942.

A nonhemolytic streptococcus meningitis developed in a 7 month old boy after a slight nasal cold; after eleven days of treatment with sulfanilamide, the spinal fluid became normal and the child seemed well again. Eleven days later the child had a relapse, and only after five and a half weeks was the spinal fluid again normal. The dose of sulfanilamide given in the first attack was 25 cg. per kilogram of body weight, and in the relapse 40 cg.; during the relapse an intraspinal injection of sulfanilamide was given also.

HIGGINS, Boston. [AM. J. DIS. CHILD.]

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND THE PHILADELPHIA LARYNGOLOGICAL SOCIETY

GABRIEL TUCKER, M.D., *Chairman*
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GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Regular Meeting, Dec. 15, 1943

Reconstruction of the Deformed Nasal Septum. A Critical Evaluation of the Orthodox Submucous Resection. Presented by DR. MATTHEW S. ERSNER.

This article will be published in full in the ARCHIVES.

DISCUSSION

DR. WARREN B. DAVIS: Dr. Ersner mentions as the indications for submucous resection (1) a septum "found pressing on the lateral nasal wall, interfering with the drainage of an infected ethmoid labyrinth or a frontal sinus"; (2) "an exostosis of the septum" which "becomes an irritant of the sphenopalatine ganglion by pressing on the posterior tip of the middle turbinate." To these I should add pressure about the anterior end of the middle turbinate which may or may not be associated with infection of the ethmoid sinus but which does produce pain of a type not infrequently erroneously classified as "hemicrania." The removal of such pressure has been a most satisfactory procedure in giving relief from this more or less persistent or frequently recurring type of pain or neuralgia.

My colleagues and I have not observed in noses which were properly operated on the symptoms from "flaccid septum" which Dr. Ersner described. The impropriety of the operations with which we have seen this condition has been that the amount of bone and cartilage removed was entirely too extensive, especially if no supporting cartilage was left at the anterior portion of the septum. It is seldom that one obtains sufficient septal cartilage without too extensive resection to correct a true saddle nose. I do agree, however, that there are many cases in which there is a moderate degree of depression of the dorsum nasi in which the insertion of septal cartilage is of definite benefit, especially in the improvement of the patient's appearance in profile. The almost complete removal of the septal cartilage, which unfortunately is still too frequently seen, may necessarily result in the drooping of the nasal tip and possibly some degree of retraction of the columella.

I cannot agree with Dr. Ersner in his statement that the development of a saddle nose following submucous resection is due to the type of attachment of the upper lateral cartilage and not to the amount of cartilage and bone removed in the operative procedure. I believe that the probability of the development of a saddle nose following a submucous resection is in direct proportion to the amount of bone and cartilage removed and that it is influenced also by the age of the patient, the younger patients being much more apt to show a saddle nose following the submucous resection than those well past the age of adolescence. In my own practice at least 50 per cent of the patients who have come for correction of a saddle nose of this type have come from the practice of one man and his staff associates. The technic of this group is such that an unusually—and I believe unnecessarily—large amount of bone and cartilage is removed. Certainly I do not believe that his series of patients would include a larger group with long upper lateral cartilage attachments than those from any other equally extensive hospital service.

May I quote from a paper, "Some External Nasal Deformities and Methods Used in Their Repair," which I read at the annual meeting of the American Laryngological Association in 1942? "The nose is such a conspicuous feature that its deformities and their corrections naturally arouse an appreciable general interest. Recently, however, that interest has been unduly intensified by ill-advised articles on plastic surgery in some lay magazines and in the Sunday supplements of some metropolitan newspapers. Thus the public has become quite 'nose-conscious' and I fear that in too many instances emphasis has been placed upon the repair of insignificant deformities in hypersensitive introspective individuals. I refer especially to minor abnormalities in the contour of the alar cartilages which, after operative procedures by insufficiently skilled surgeons, have been converted not infrequently into actual deformities of varying degrees, thus giving such patients ample justification for remaining in the dissatisfied throng. This situation needs to be combated more efficiently by ethical plastic and reconstructive surgeons. Real nasal deformities, however, are numerous, the problems encountered in their correction are intensely interesting, and in the hands of properly trained reconstructive surgeons the functional as well as the cosmetic results are usually gratifying both to the patient and to the surgeon."

I still believe that there is too much operative work being done on insignificant deformities of the alar cartilages and the nasal tip. One must be exceedingly careful in selecting the patient on whom these operations should be done. In many instances one sees results with which a patient is justified in being dissatisfied; there are others in which the patient may show slight improvement in appearance but is still dissatisfied and has possibly been made more introspective by the operative procedure.

I fear that accurate operations on the alar cartilages are not as simple as one might infer from the papers which the members have heard. In those cases in which operations are definitely indicated, it is wise to avoid carrying the incision through the free anteroinferior edge of the lower lateral cartilage, since when the division is complete the subsequent contour of the nostril is seldom perfect. The correction of the drooping nasal tip is not so difficult, and in most instances any excision of the deviated anteroinferior edge of the septal cartilage can be readily done at the same time.

My plea is for (1) greater care at the time of operation to avoid perforation of the septum, (2) resection of the minimal amount of septal cartilage and bone which will correct the deformity, (3) avoidance of operations on slight irregularities in alar cartilages, especially in

urotic persons and (4) use of conservative procedures all operative rhinoplastic cases.

Dr. George M. Coates: I take it Dr. Ersner's paper concerned chiefly with the physiologic effect of operaons on the nose, the cosmetic effect being incidental. Dr. Ersner said, in some of these cases the rhinolot may not get a good result because the upper lateral rtilages have some deformity which may not have en taken into consideration when the submucous rection was performed.

It is not necessary to have all the air on one side ing to the upper part of the nose. One must examine e nose inferiorly also in these cases. The pictures ewed here are perfectly familiar to all rhinologists. e does a submucous resection and neglects to do ything more.

Rhinologists will all have to take into consideration e importance of removal of the anterior thirds of iddle turbinates as well as the relation of these septal viations to the external deformities which are occanally seen.

It seems to me that in surgical treatment the keynote to reconstruct externally when this is indicated and remove a thickened deflected septum when necessary. is true that there is always a great temptation to ke out. As Dr. Poretz says, the air comes in over ges and spurs. The bony septum may sometimes ake little difference. One has occasionally seen a disrted columella, a saddle back nose or a depressed se following an operation which possibly was done the operator in his eagerness to give the patient a od airway.

Dr. Saks Bricker: Dr. Ersner is to be commended his paper. There are, however, several points of his scussion that should be commented on. Saddle nose llowing a submucous resection is due to a number of her factors more important than those of a long or a ort triangular cartilage. It is more frequently a matter surgical technic. Removal of the quadrilateral carage too high, a cartilage that is congenitally thin or ft, or a dislodgment of the upper part of the quadriteral cartilage when using the Ballenger knife are a w of the contributing causes of a postoperative weakns of the bridge of the nose.

No submucous resection can give satisfactory results the nares are obstructed and narrowed so as to prent ingress of sufficient air on inspiration. A lateral ring of the internal cruri of the alar cartilages must o be considered. Paresis of the alar cartilages causing llapse of the nostrils on inspiration can be corrected the removal of an elliptic bit of tissue from the ercartilaginous area and the suturing together of e triangular and alar cartilages, thereby tightening the eral sides of the nose. A columella which is too wide too short should be narrowed or lengthened. Elevans of mucosa on the floor of the nose which improply divert the column of inspired air should be removed. there is a necessity for removing the quadrilateral rtilage anteriorly and thereby weakening the tip, then e columella should be split and a strut of cartilage ced between the anterior nasal spine of the maxilla d the tip of the nose. An operation on the septum t does not allow as straight a column of air as posle to enter the nose cannot give a satisfactory result.

Dr. Henry Miller: Dr. Ersner's paper is valuable many respects. I think he talked about limitations of e orthodox submucous resection. As all know, the pe of deviation for which it is used is the usual type t follows trauma in childhood. Metzenbaum brought s out some time ago.

I enjoyed the paper. I think that there is a growing recognition of rhinoplastic surgery.

Dr. Ersner: I wish to take this opportunity to thank Drs. Davis, Coates and Bricker for their kind and constructive discussion.

The purpose of my paper was not to stress technic but rather to emphasize the importance of reestablishing nasal physiologic function by reconstructive surgery.

My plea to the rhinologist was not meant for those who are not prepared to do nasal reconstruction. It was my aim to encourage the recognized rhinologist to assume full responsibility for the nose, both internal and external. In my opinion rhinologists may be witnessing the evolution of a new school of thought, with the idea that the submucous resection in some cases is not limited to the septum but may be rather a procedure of reconstruction in which one must take into consideration the nose in its entirety.

Dr. Coates questioned my account of the saddle nose following submucous resection. I should like to reemphasize the importance to me of the short upper lateral cartilages in their relationship to the quadrangular cartilage. In my opinion it is the shortness of the upper lateral cartilages rather than the complete removal of the ventral end of the septal cartilage which is responsible for the saddle nose. I hope that this will answer Dr. Coates's question.

One of the phylogenetic characteristics of the Negro is the flat nose. I examined 60 negroid noses to satisfy my curiosity, and I found that in most instances they had short upper lateral cartilages. I might add that when any nose is characterized by short upper lateral cartilages that nose is a potential saddle nose when a submucous resection is to be performed.

I am indeed happy that the discussers have agreed with me in the principle that the nose in its entirety is the responsibility of the rhinologist and that the specialty of the rhinologist should not be limited to intranasal surgery but should include also the reconstruction, when necessary, of the columella, the upper and lower lateral cartilages and the bony framework.

The First Fifty Years of the Section on Otolaryngology of the College of Physicians. Presented by Dr. George B. Wood.

In having been asked to prepare a review of the origin and activities of the Section on Otolaryngology of the College of Physicians I feel myself greatly and also unduly honored. This is the fiftieth anniversary of our section. At the time of its inception there were in this city no societies devoted to our specialty. The organization started by J. Solis-Cohen in 1881 and known as the Philadelphia Laryngological Society had ceased functioning in about 1887. However, about 1911 the Philadelphia Laryngological Society was reorganized and has had an active career ever since. I desire at this time to pay tribute to the important part it has played in the advancement of otolaryngology. However, tonight I speak only of our section, as this is its anniversary that we are celebrating. It might have been of interest to have interpolated into this history of our section short biographic sketches of the more prominent pioneers. However, there is not the time or the space for such biographies. For the majority of us, mere mention of their names is sufficient to make us realize that giants started our section and steered its early course.

The story of the beginning of the section and its continued activity from the time of its formation to the present day is closely associated with the rise and progress of our specialty in Philadelphia. At the time

of its organization the need for specialization was being more and more recognized, although there were no systematic courses for specialized training, and every physician who desired to specialize had to work out his own salvation in clinic and laboratory. In spite of these handicaps there developed a group of men in Philadelphia whose names have been handed down to posterity as truly great otolaryngologists. It is natural that such men, believing in the more complete development of otolaryngology as a special branch of medicine, should feel the necessity of such an opportunity for the pooling of ideas and clinical experiences as would be afforded by regular meetings entirely devoted to this subject.

As the membership of the College of Physicians at that time included the leading exponents of our specialty, the teachers in the medical schools and the chiefs of the otolaryngologic departments of the various hospitals, it was logical and proper that the desired meetings should be a part of that institution, i. e., that a section of the college be organized and devoted to the study of and the promulgation of the various subjects of otolaryngology.

Chapter XIII, section 1, of the By-Laws of the College of Physicians states that "Whenever fifteen Fellows of the College shall, in writing, certify to the President their desire to have organized a Section upon some department of medical science or practice, he shall, if in his judgment it seems wise, direct the Secretary of the College to announce the creation of such a section." Therefore, there was sent to the president in the fall of 1893 a petition asking for the organizing of such a section. It was signed by J. Solis-Cohen, Harrison Allen, George C. Harlan, Samuel D. Risley, Alexander W. MacCoy, Ralph W. Seiss, S. Solis-Cohen, Louis Jurist, Eugene Vansant, Charles Schauffner, Walter J. Freeman, Joseph S. Gibb, Frank Woodbury, B. Alexander Randall and F. M. Perkins. On Dec. 12, 1893 Dr. S. Weir Mitchell, then president of the College of Physicians, appointed as executive committee of said section, J. Solis-Cohen, Charles H. Burnett and B. Alexander Randall.

The first meeting of the section was held Dec. 20, 1893, in the hall of the College of Physicians, at Thirteenth and Locut Streets. There were present: J. Solis-Cohen, Harrison Allen, Alexander W. McCoy, S. Solis-Cohen, Louis Jurist, Ralph W. Seiss, Walter J. Freeman, Charles Schauffner, Howard F. Hansell, Joseph S. Gibb and B. Alexander Randall. There were also present, as invited guests, A. H. Cleveland, George C. Stout and Barton H. Potts. Dr. Harrison Allen was elected chairman and Dr. Burnett, clerk. After organization, the scientific part of the meeting included a demonstration by Dr. Randall of the method of making casts of the nasal sinuses, the pharynx, the larynx, the bronchi and the auditory canals by the galvanic coating of copper over the original cast. Dr. J. Solis-Cohen reported the removal of a sarcoma through the mouth, with no sign of recurrence "after months," and in the discussion of this paper Dr. McCoy referred to "a case of malignancy" too extensive for operation which was treated by the injection of methyl rosaniline chloride U. S. P. with disappearance of the mass and of the associated enlargement of the lymph nodes.

The business part of the meeting included a resolution concerning the date of future meetings; the first Tuesday of the month was preferred, but the third Wednesday was voted as an alternative date. There was also a debate as to the wisdom of employing a stenographer to report the discussions, but it was decided "that members could reduce to writing their own remarks."

At the time of the formation of our section there was apparent a certain exclusive pride in being a Fellow of the College that led to our restricting, which happily was of short duration. The first Card of Notice for a meeting was that of Feb. 6, 1894, and this card was similar to those used for many years except that it had printed at the bottom:

"A part of Section Six of the Rules of the College governing Sections to wit:

"No person not a Fellow of the College shall attend any meeting of the Section except by specific invitation to that meeting by a Fellow who shall give the name of the visitor to the Clerk of the Section and be responsible for the visitor's actions at the meeting."

However, in spite of this restriction there were present at this first meeting as invited guests Dr. Ring, Dr. Hammond, Dr. Rowan and Dr. Cleveland. I suppose the responsibility for the visitors' actions was more than the members could bear, because this particular paragraph was omitted from the Card of Notice of Dec. 11, 1894 and never again reinstated. However the names of guests were faithfully recorded in the minutes of the meetings until November 1900.

It might be of interest to note the various subjects that were taken up at these early meetings, and one finds that many of the questions that are discussed today were being discussed by specialists in those days. For instance, infectious diseases of the middle ear with complicating mastoid, intracranial and labyrinthian involvements occupied an important space in the meetings. There was one quite significant paper by Burnett, "The Synopsis of Twenty-Six Cases of Chronic Tympanic Vertigo and Tinnitus Relieved by Intratympanic Operations." Then there were many papers on malignant neoplasms; on anatomy and histology; on the technic of intranasal operation; some on foreign bodies in the nose and esophagus, and so on. At the meeting held on Feb. 6, 1894 a case of cerebral abscess with recovery was reported by Dr. Randall, and another similar case was reported by Dr. Burnett—the patient operated on by Dr. DeForest Willard—also with recovery. In 1899 Dr. Vansant reported on "The Use of Dried Extract of Suprarenal Capsule in Nasal Surgery." This was one of the earliest communications concerning that important drug.

In 1925 it was decided to replace the regular May scientific meeting with a dinner limited to the Fellows of the Section on Otolaryngology and to the social enjoyment of each other's company. That year it was held at the Art Club. While this festivity was repeated a few times, it did not become a regular feature.

There is no need for me to review the further progress and activities of our section through the recent years. You of the audience are responsible for its achievements, and your success in carrying on is self evident. You now boast an enrolment of 56 Fellows and there has been established throughout the country a reputation collectively and individually that makes us proud and happy. Again, as in 1918, many of our Fellows have given up their active practices to risk their lives and future in the service of their country. Let us pause for a moment as we salute these of our fellowship: Edward H. Campbell, C. E. Cornelius, Robert J. Hunter, Harry P. Schenck, Louis E. Silcox and Austin T. Smith.

Because of the present war we have found it necessary to curtail some of our activities. We have joined with the Philadelphia Laryngological Society in combined meetings, thus saving needed hours for other purposes and adding interesting and instructive experiences to the meetings. We of the section are glad of this oppor-

nity to meet with our fellow otolaryngologists in mradeship and in debate and discussion.

The Section on Otolaryngology has the heritage of eat and progressive achievement, and this is a stimulus at will greatly help us to carry on through these days world disruption. Our minds are occupied with the tails of the human struggle for right and freedom; r souls are numbed by anxiety and sorrow and our ys filled with an excessive burden of professional ork; but we shall find time and interest to keep the tivities of this section alive and virile. As we look rward to the day when strife shall have ceased and aceful pursuits shall once more be the predominating otif of living, it is on you of the younger generations nt the responsibility of our section's future progress ust lie.

DISCUSSION

DR. WALTER ROBERTS: When Dr. Wood found my me on one of the papers presented before this section the College of Physicians in 1899, he jumped to the nclusion that I must be one of the "old fellows" and asked me to reminisce a little. I do not remember presenting such a paper but if the records show that, it must be correct. In those days our meetings were held at the Old College of Physicians, located at Thirteenth and Locust Streets. We had a small meeting room, about 12 by 15 feet square. The membership was about 15, and usually 8 or 10 members were present. The first chairman was Dr. Harrison Allen. I was not then a member of the section, and, although I had heard very much about Dr. Allen, I did not know him personally.

Dr. Ralph W. Seiss, one of the early members, was a tall, smooth-shaven gentleman—always rather stylishly clad. He had an impressive manner of speaking and had a great respect for his own opinions. I got along rather well with him because I made it a point never to argue. In spite of his conceit he had a large practice among the wealthy people in Philadelphia. He did no operating, but he had a gentle touch and was most skilful in giving pleasant local treatments. He had a wonderful collection of sprays for eliminating all kinds of local pathologic conditions. When he was sick, I took care of his practice and greatly enjoyed the experience.

Book Reviews

tosclerosis, Volume IV (1936-1942 Inc.) and Future Loose Leaf Additions. An index of the literature with abstracts and an index of authors according to subjects (year by year). Edmund Prince Fowler. Issued by the Central Bureau of Research of the American Otological Society, Inc. Price $3.50. New York: Central Bureau of Research, 2 East One Hundred and Third Street, 1944.

With "Otosclerosis, Volume IV," the editor has hanged from the sewn leaf of former volumes to a ose leaf so that the contributions to the literature in e future may be inserted in the cover yearly, thus vercoming the former lag in publishing the index. It the intention now to keep it up to date year by year, ssen the cost of publication, secure a wider distribution nd thereby increase the usefulness of the volume.

There may be some criticism because of the curtailent of the bibliography in volume 4, but this probably uld not be avoided, owing to the dislocations occaioned by the war, and, as the editor says in his prefce, "tardily received manuscripts will be printed in e subsequent loose-leaf installments so that they may e inserted in the binder in their proper order." Another riticism may be that some of the more important conributions are not abstracted at greater length. This o doubt will be given careful thought, although here e personal element cannot help but influence somehat the choice of articles to be so abstracted. Howver, there would appear to be no bias in these matters.

Some repetition necessarily occurs, but this is unvoidable with a subject like otosclerosis and so many apers containing previously published facts and ideas.

Considering the war shortages, the publishers have een fortunate in procuring such excellent binder and ress work. The names of the authors stand out in old relief as they should in any index, and the type sed in the titles and abstracts is pleasing to the eye nd easily read.

It is certain that the editor would appreciate receiving rom all of those who consult this volume any conructive criticism they would be good enough to make. It is hoped that this yearly index with abstracts will be continued indefinitely. It supplies a ready and complete reference to the literature not elsewhere obtainable.

Clinical Audiometry. C. C. Bunch. Price $4. St. Louis: The C. V. Mosby Company, 1943.

Perhaps no one in the country was better fitted to write on clinical audiometry than was the author of this book, lately deceased. Though the book was published posthumously, it is a most complete text. There was and will be for some time a great need, and it is to be hoped a great demand, for it from otologists. It ties in the present with the past, preserving what is of value in the old testing methods and explaining the audiometric advances of recent years.

The author is clear and direct in his descriptions of tests and in his analysis of findings. Technics are carefully given. Malingering is clearly but briefly handled. There are excellent chapters on sound-proofing and on the development of modern audiometry—two subjects rarely found discussed in textbooks.

Under audiometric findings one cannot but be convinced that there are still great uncertainties in making a diagnosis on an audiogram alone. Yet audiometry has made many of the old concepts of the location of the hearing loss untenable. This is particularly true as to the loss of high tones, which were supposedly well retained in catarrhal deafness.

The little understood subjects of masking and auditory attention are carefully described.

The chapters on hearing aids, the use of residual hearing and the deaf child deal with material unfamiliar, unfortunately, to many otologists.

The book is replete with well selected illustrations, which further clarify the text. There is little omitted from the complete picture of the subject, yet the book does not seem congested or labored, and is certainly "easy reading."

All in all, here is a fitting memorial to a truly industrious, careful physiologist who, working continuously in ear clinics, has made invaluable contributions to our specialty.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Chicago. Time: June 12-16, 1944.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.
President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: New York. Time: June 6, 1944.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th S New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bl St. Louis.
Place: New York. Time: June 7-8, 1944.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. H. Marshall Taylor, 111 W. Adams S Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical A Bldg., Rochester, N. Y.
Place: New York. Time: June 9-10, 1944.

SECTIONS:

Eastern.—Vice President: Dr. Westley M. Hunt, 90 5th Ave., New York 21.
Southern.—Vice President: Dr. William C. Warren J 478 Peachtree St. N. E., Atlanta, Ga.
Middle.—Vice President: Dr. Fred W. Dixon, 2060 9th St., Cleveland.
Western.—Vice President: Dr. Simon Jesberg, 500 Lucas Ave., Los Angeles.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st S New York 21.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., N York.
Place: New York. Time: June 5-6, 1944.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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